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Table of Contents.

CARDIAC INFARCTION AND CORONARY DISEASE IN GENERAL IN POST-MORTEM EXAMINATIONS.[1]

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THIS paper is based on a series of 6000 tabulated post-mortem examinations carried out at the Royal Adelaide Hospital between the years 1920 and 1946, together with the results from 600 autopsies at the mental hospitals in South Australia, and 250 autopsies performed for the coroner during the war years.

The findings have been arranged under the following headings: (i) cardiac infarction, when there was obvious recent necrosis of cardiac muscle; (ii) fibrosis of the cardiac muscle, when muscle substance had been replaced by fibrous tissue, either as a result of infarction, from which recovery had occurred, or as a result of the slow silting up of the coronary vessels interfering gradually with the vascular supply to portions of the muscle; (iii) coronary disease of cardiac importance, comprising a number of cases in which the atheromatous changes were so considerable or the clinical course of the case was such that there was good reason to consider that the coronary disease had played an important part in the illness and death of the subject. It may be assumed that in nearly every instance where infarction was found, the patient had died in some way as a result of that condition, although there were occasionally instances in which death was really due to some unrelated condition. In a number of the cases of cardiac fibrosis the patient had died from unrelated conditions. The same applies to the examples of coronary disease of cardiac importance.

[1] Read at a meeting of the Section of Pathology, Bacteriology, Biochemistry and Experimental Medicine, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

Cardiac Infarction.

Of the 147 cases of cardiac infarction (Table I), 125 occurred in the course of a series of 6000 tabulated post-mortem examinations made at the Royal Adelaide Hospital between the years 1920 and 1946. Ten cases occurred in 600 autopsies at a mental hospital, and 12 in the course of 250 recent coroner's cases. Cardiac infarction is typically due to thrombosis occurring in the course of atheroma. Thrombi may form as a result of the projection of the atheromatous patch in the vessel wall on which platelets may be deposited followed by the formation of a thrombus. This condition may be facilitated by the occurrence of small hæmorrhages from capillaries in the deeper part of the atheromatous area. Probably actual thrombosis is not always necessary for the occurrence of infarction, as in some cases no actual thrombosis could be detected, though there was much atheroma. It is possible that in some instances spasm of a vessel may alone by responsible for this condition, and probably spasm in a vessel with atheromatous patches would be specially prone to interfere with the nutrition of the muscle.

In the successive 6000 autopsies, the incidence of infarction of the heart muscle was as follows: 11, 5, 11, 28, 25, 45 (total 125). It should be noted that there was a sudden increase in the fourth thousand autopsies, which were commenced in 1935 and were completed early in 1940, and that there has been another sudden jump in the sixth thousand autopsies, which began early in 1943 and ended in 1946. The number of cases is far beyond that which might be accounted for by aging of the population.

There were 95 instances of cardiac infarction in men and 52 in women in the total of 147 (see Table I). It will be seen that for sudden deaths in coroner's cases, the figures, though small, show a decided preponderance among men (10 to 2), though sex would make no difference in the necessity for such post-mortem examinations. Males also preponderate slightly in mental hospital cases (6 to 4). In the Royal Adelaide Hospital series there were 79 males and 46 females in 125 subjects; as at the Royal Adelaide

TABLE I.

Cardiac Infarction: 147 Cases.

Source of Cases.	Age Group (Years).								Total.
	Twenties.	Thirties.	Forties.	Fifties.	Sixties.	Seventies.	Eighties.	Age Unknown.	
Male Subjects.									
Royal Adelaide Hospital:									
First thousand	—	—	1	—	2	3	2	—	8
Second thousand	—	—	—	1	1	2	—	—	4
Third thousand	—	—	2	1	2	—	1	—	6
Fourth thousand	—	—	1	3	9	3	—	—	16
Fifth thousand	—	—	2	2	3	5	1	—	13
Sixth thousand	—	—	4	6	14	6	2	—	32
Mental hospitals: 600 post-mortem examinations	—	—	1	1	3	—	—	1	6
Coroner's cases: 250 post-mortem examinations	—	1	2	1	2	4	—	—	10
Totals	—	1	13	15	36	23	6	1	95
Female Subjects.									
Royal Adelaide Hospital:									
First thousand	—	1	—	—	1	1	—	—	3
Second thousand	—	—	—	1	—	—	—	—	1
Third thousand	—	—	—	2	1	2	—	—	5
Fourth thousand	—	—	1	2	6	2	1	—	12
Fifth thousand	1	—	—	3	7	1	—	—	12
Sixth thousand	—	—	—	3	6	4	—	—	13
Mental hospitals: 600 post-mortem examinations	—	—	—	1	2	—	1	—	4
Coroner's cases: 250 post-mortem examinations	—	—	1	—	—	1	—	—	2
Totals	1	1	2	12	23	11	2	—	52
Grand total	1	2	15	27	59	34	8	1	147

Hospital approximately two autopsies are performed on men to one on a woman, it will be seen that here the incidence is slightly greater among women than among men, as there should have been about 84 males to 42 females.

In the seventh thousand autopsies at the Royal Adelaide Hospital, 800 had been completed by the beginning of July, 1948. There were 34 cases of cardiac infarction, 23 in men and 11 in women. The incidence seems to be at least as great as in the sixth thousand. Two of the males were in the forties.

Even relatively young people may die from this condition, as one woman was aged twenty-eight years and another thirty-eight years, and a man was aged thirty years. There were fifteen subjects in the forties. However, the chief incidence in both sexes is in the sixties, with considerable numbers in the fifties and seventies.

Hypertrophied Hearts with Cardiac Infarction.

During the last 3000 post-mortem examinations at the Royal Adelaide Hospital, 52 out of 98 subjects of cardiac infarction (that is, more than half) had hypertrophied hearts without adequate valvular or renal cause. Forty out of 61 male subjects (that is, two-thirds) had this hypertrophy, but only 12 out of 37 female subjects (that is, one-third).

Several cases of cardiac infarction with aortic stenosis or mitral stenosis and hypertrophied hearts also occurred, so it would appear that it was the hypertrophy of the heart, rather than the high tension, that was the responsible factor in this tendency for infarction to occur.

Seventeen at least out of these 52 hearts (all were not weighed) had weights over 20 ounces (568 grammes), sixteen of them from males, one from a woman. These weights were 28 ounces (800 grammes), 25 ounces (710 grammes) (this specimen had ante-mortem clot in the cardiac chamber), 24 ounces (680 grammes) (two hearts), 23 ounces (652 grammes) (three hearts), 22 ounces (622 grammes) (three hearts), 21 ounces (593 grammes) (two hearts, one from a female), and 20 ounces (564 grammes) (five hearts).

Rupture of the Infarcted Heart Muscle.

There were 24 cases of rupture of or leakage through the infarcted heart muscle in the 147 cases. Twelve of these occurred in 125 infarctions found at 6000 post-mortem examinations at the Royal Adelaide Hospital, four in ten infarctions at 600 such examinations at the mental hospitals, and eight in twelve infarctions at 250 autopsies performed for the coroner.

Of the 24 subjects, 13 were males and 11 females. The ages of the males were as follows: forty-seven years, three in the sixties, eight in the seventies, eighty-seven years—a total of 13. The ages of the females were: fifty-eight years, seven in the sixties, two in the seventies, age of one unknown—a total of 11.

Of the twelve subjects at the Royal Adelaide Hospital, six were men and six women; of the four subjects at the mental hospitals all were women; and in the eight coroner's cases, seven subjects were men and one was a woman.

The amount of blood and clot, in ounces, present in the pericardial cavity was recorded in seven cases at the Royal

Adelaide Hospital as follows: 26, 23, 12, 10 (three cases) and 8. In the coroner's cases the amounts in ounces were 5, 18, 13, 14, 16, 16, 17 and 18; and in the mental hospital cases the amounts were six ounces of serum and clot, eight ounces of clot and serum, eight ounces and ten ounces.

Comment.—It has been noted by others that rupture of the heart from cardiac infaretion was more likely to occur in patients in mental hospitals than in those in general hospitals. The inference seemed to be that the insane as such were more predisposed to this accident than the mentally healthy. The above figures suggest that this is a fallacy. It will be seen that whilst about one in ten subjects of cardiac infarction in a general hospital die from rupture, nearly half (four out of ten) of those in mental hospitals may do so, and more than half the persons dying suddenly or found dead, and coming under the notice of the coroner and having cardiac infarction, die suddenly from rupture. Though the figures are small, they seem to show that the insane are behaving in this respect like the populace at large. There are a number of cases of cardiac infarction which are silent as regards symptoms; the patient does not complain or at any rate does not seek medical advice. The mere fact that examination of the cardiac muscle shows necrosis indicates that the interference with the blood supply has been present for an appreciable number of hours, if not days. Such muscle is obviously likely to rupture or leak, and when it does so death is so rapid that the patient usually dies before reaching hospital or receiving medical attention, and consequently comes under the jurisdiction of the coroner, or in the case of mental hospital patients, the sudden death makes a post-mortem examination inevitable. Patients already in a general hospital for their cardiac or some other condition are more likely to be at rest, and death is more likely to occur from gradual cardiac failure or other cause than from rupture or leakage through the infarcted muscle.

Ante-Mortem Thromboses in the Heart in Cardiac Infarction.

In the cases of cardiac infarction in the last 3000 autopsies, there were 32 instances of ante-mortem clots in the chambers of the heart, 21 in males, 10 in females, and one in a subject whose sex was not noted.

Of the 32 subjects, 21 had clots in the left ventricle only, one had clots in the left ventricle and left auricle, four had clots in the left ventricle and right auricle, one had clots in the left ventricle and both auricles, four had clots in both ventricles, and one had clots in the right auricle only.

In one instance the clot nearly filled the left ventricle. In two instances a ball thrombus was nearly free in the left chamber, and in another a ball thrombus was in the right auricle.

Myocardial Fibrosis from Coronary Disease.

There were 92 instances of appreciable myocardial fibrosis in the last 3000 tabulated post-mortem examinations at the Royal Adelaide Hospital, 77 in men and 15 in women (Table II).

Of the seventh thousand autopsies, 800 had been performed by July, and in these there were 31 instances of fibrosis, 23 in men and 8 in women, the youngest man being aged twenty-one years and having a greatly hypertrophied heart. The chief incidence is in the sixties and the next highest in the seventies, closely followed by the fifties.

In the fourth thousand, 12 out of 13 subjects with fibrosis in the heart muscle died from cardiac causes, though the chief factor responsible may have been failure of the hypertrophied heart so often associated with the fibrosis. Nevertheless, the fibrosis probably played a prominent role. In the fifth thousand 25 of the 38 subjects, and in the sixth thousand 22 of the 41 subjects, so died. Of the last, it may be noted that 17 of the 22 with hypertrophied hearts so died, but only five of the 19 with cardiac hypertrophy. Of the remaining five of the 22 with hypertrophied hearts, a woman, aged sixty-five years, died from massive pulmonary embolism, and a man, aged fifty-one years, from bacterial endocarditis.

Two deaths occurred in the casualty room—that of a man, aged sixty-two years, whose heart weighed 18 ounces (512 grammes), and a man, aged sixty-eight years, whose heart was not hypertrophied. A man, aged seventy-seven years, without cardiac hypertrophy, had an aneurysmal bulge filled with ante-mortem clot in the posterior wall of the left ventricle.

In the last 3000 autopsies, 40 out of 92 subjects of cardiac fibrosis from coronary disease had hypertrophied hearts. Only seven of the 40 were females. Eleven of the subjects (all males) had hearts weighing more than 20 ounces (568 grammes), of which one weighed 28·5 ounces (808 grammes), one 27 ounces (760 grammes), one 25·5 ounces (722 grammes) and two 24 ounces (683 grammes).

Aneurysmal Dilatation of the Wall of the Heart.

Aneurysmal dilatation of the degenerated or fibrosed wall of the heart occurred in five males in the Royal Adelaide Hospital series, whose ages were sixty-eight, seventy-four, seventy-five, seventy-seven and seventy-eight years respectively. In the man of seventy-five years the bulge had ruptured.

TABLE II.

Myocardial Fibrosis from Coronary Disease.

Subjects.	Age Group (Years).						Total.
	Thirties.	Forties.	Fifties.	Sixties.	Seventies.	Eighties.	
Males:							
Fourth thousand ..	—	—	4	5	2	—	11
Fifth thousand ..	—	2	4	13	9	4	32
Sixth thousand ..	—	3	9	9	11	2	34
Totals	—	5	17	27	22	6	77
Females:							
Fourth thousand ..	1	—	—	1	—	—	2
Fifth thousand ..	—	—	4	—	2	—	6
Sixth thousand ..	—	—	—	5	1	1	7
Totals	1	—	4	6	3	1	15
Grand total	1	5	21	33	25	7	92

Rupture of a Papillary Muscle.

In a woman, aged fifty-seven years, a papillary muscle had ruptured and the free flap had passed through the mitral valve, suggesting regurgitation.

Ante-Mortem Thromboses in the Heart in Cardiac Fibrosis.

In the 92 cases of cardiac fibrosis in the last 3000 autopsies, there were fourteen instances of ante-mortem clots in the chambers of the heart (eleven among males, three among females).

Of the fourteen subjects, nine had clots in the left ventricle only, three had clots in the left ventricle and right auricle, one had clots in the left ventricle and both auricles, and one had clots in both auricles.

One of the nine with clots in the left ventricle, a male subject, aged seventy-four years, had an aneurysm of the fibrosed patch in the ventricle.

Coronary Atheroma of Cardiac Importance.

During the last 3000 autopsies at the Royal Adelaide Hospital, a category under the caption "Coronary Atheroma of Cardiac Importance" (Table III) has been employed to include cases in which there was considerable or at least

appreciable atheroma, in places with more or less narrowing of the lumina of the coronary vessels, but no obvious recent necrosis of muscle (infarction) or fibrosis, in which the clinical history and the absence of other causes for death seemed to show that the coronary disease was probably responsible for the fatal result, or contributed to it. A number of coroner's cases in which persons are found dead or die suddenly come under this heading. A few of these cases may, of course, be due to infarction which is not yet old enough to produce a naked-eye or even microscopic change, and in which no clot is detected to give a clue to what has happened.

Under this heading appear 106 cases, 80 in males and 26 in females (Table III):

TABLE III.

Coronary Atheroma of Cardiac Importance.

Subjects.	Age Group (Years).							Total.
	Twenties.	Thirties.	Forties.	Fifties.	Sixties.	Seventies.	Eighties.	
Males:								
Fourth thousand	—	—	—	2	8	2	1	13
Fifth thousand	1	—	1	4	12	9	7	34
Sixth thousand	—	1	5	9	6	8	4	33
Totals	1	1	6	15	26	19	12	80
Females:								
Fourth thousand	—	—	—	3	2	2	—	7
Fifth thousand	—	—	—	1	3	5	1	10
Sixth thousand	—	—	—	1	4	3	1	9
Totals	—	—	—	5	9	10	2	26
Grand totals ..	1	1	6	20	35	29	14	106

The accompanying circumstances suggest that about 63 of these 106 subjects died as a result of their coronary disease. It is interesting to note that 33 of the 63 had hypertrophied hearts. In the fourth thousand, 16 out of 20 subjects probably died from the coronary disease, four having hypertrophied hearts; in the fifth thousand, 30 out of 44 subjects probably died from the coronary disease, 15 having hypertrophied hearts; in the sixth thousand, 17 out of 42 subjects probably died from coronary disease, 14 having hypertrophied hearts. This makes a total of 63 out of 106 subjects, and a total of 33 hypertrophied hearts. Ten of these hypertrophied hearts weighed over 20 ounces (564 grammes), and 18 at least over 18 ounces (510 grammes). In 15 of the 18, the hypertrophy had apparently been due to hyperpiesis; one of these hearts weighed 28 ounces (808 grammes) and another 25·5 ounces (712 grammes). In two cases the hypertrophy was apparently due to aortic stenosis with calcification (weights 22·25 ounces or 625 grammes and 21·25 ounces or 600 grammes). In the remaining case the hypertrophy seemed to have been due to the high tension of red granular kidneys (weight 21 ounces or 593 grammes).

One patient in the fifth thousand and three in the sixth thousand had collapsed and died suddenly, and probably others did so likewise, but, as they were in hospital, this fact was not recorded in our data. Two in the fifth thousand died under anæsthetics.

Sixty-Six Coroner's Cases of Sudden Death Attributed to Coronary Disease or its Effects.

In 66 cases the coroner had ordered post-mortem examinations on account of the person's being found dead or dying suddenly without having been under recent medical supervision. The number of coroner's cases from all causes was 250, including murders and suicides, so that these 66 cases comprise more than a quarter of the total. I have grouped them according to whether there was infarction of the heart muscle with obvious recent necrosis, or fibrosis of the muscle due to a gradual silting up of the branches of the coronary vessels or to repair of an infarcted area, or atheromatous coronary disease without these consequences.

It should be noted that all these persons were found dead or had died suddenly, and that the cause of death as far as could be ascertained by the post-mortem examination was disease of the coronary vessels. Cardiac infarction was present in twelve cases, in eight of which rupture of the infarcted area or leakage through it had also occurred. The youngest of the subjects of cardiac infarction was a man, aged thirty years, who was found dead in bed, and the next youngest was a woman, aged forty-six years, who was found dead on her bed during the daytime; two men, aged forty-seven years, had collapsed and rapidly died, one with rupture of the infarcted area. Ante-mortem

TABLE IV.

Sixty-six Coroner's Cases of Sudden Deaths from Coronary Disease in Various Forms.

Condition.	Number of Subjects.	Sex; Age in Years.	Observations.
Cardiac infarction.	4	M., 30	Found dead in bed.
		F., 46	Found dead on her bed in daytime.
		M., 47	Collapsed at work and died at once.
		M. 53	Found dead in bed.
Cardiac infarction with rupture of the heart.	8	M., 47	Collapsed and died.
		M., 65	Collapsed and died at work.
		M., 67	Felt faint at work and died.
		F., 70	Found dead beside telephone.
		M., 71	Found dead beside wireless set.
		M., 73	Found dead in lavatory.
		M., 74	Collapsed in street.
		M., 77	Found dead in lavatory.
Atheroma with thrombi in the coronary vessels and fibrosis of the muscle.	4	M., 48	Collapsed at work.
		M., 52	Collapsed and died in two minutes.
		M., 61	Collapsed outside and died.
		M., 72	Found dead in street.
Atheroma with thrombi in the coronary vessels. Hypertrophied heart.	2	F., 47	Died suddenly at home.
		M., 48	Fell down in street and died.
Atheroma with thrombi in the coronary vessels.	2	M., 51	Felt queer all day, collapsed and died.
		M., 73	Collapsed and died.
Coronary atheroma with fibrosis of the muscle.	14	M., 42	Collapsed at work and died.
		M., 48	Collapsed and died at railway station.
		M., 52	Found dead sitting in motor bus.
		M., 54	Found dead beside his bicycle.
		M., 55	Found dead in bed.
		M., 57	Found dead in bed.
		M., 59	Collapsed and died in two minutes.
		F., 60	Found floating in sea. (? Drowned or died from coronary disease.)
		M., 61	Found dead in bed.
		M., 63	Found dead on floor of bathroom.
		M., 66	Collapsed and fell in doorway.
		M., 68	Found dead in bed.
		M., 69	Talking, turned round and fell dead.
		M., 73	Died in outhouse.
Coronary atheroma with hypertrophied heart.	13	M., 43	Collapsed and died.
		M., 46	Found dead in bed beside his wife.
		M., 53	Collapsed on railway station.
		M., 53	Found dead in bed.
		M., 54	Collapsed and died in his home.
		M., 57	Riding bicycle on platform, rode over end, died.
		M., 59	Collapsed and died immediately sitting in a lorry.
		M., 62	Collapsed on way home from work and died.
		M., 67	Died suddenly in cell at police station.
		F., 67	Died in bed, clothed.
		M., 68	Complained of pains in chest, found dead later.
		F. 69	Found dead in bed.
		M., 71	Collapsed in street at 4 a.m. and died.

TABLE IV.—*Continued.*

Sixty-six Coroner's Cases of Sudden Deaths from Coronary Disease in Various Forms.—Continued.

Condition.	Number of Subjects.	Sex; Age in Years.	Observations.
Coronary atheroma.	19	M., 35	Collapsed in bedroom and died fairly quickly.
		M., 37	Collapsed and died.
		M., 39	Trainer running to attend injured football player, collapsed, gasped and died.
		F., 43	Vomiting *et cetera* present for some hours; then died quickly.
		M., 45	Complained of pains in chest; found dead in bed.
		M., 53	Died suddenly after complaining of pain and tenderness over chest.
		M., 53	Collapsed and died at place of work.
		M., 55	Collapsed in street, died on reaching home.
		M., 56	Ran to catch a tram, collapsed and died.
		M., 57	Railway guard, collapsed and died on train.
		M., 57	Found dead in bed.
		M., 64	Found dead on floor.
		M., 64	Complained of pains in chest, went to bed, found dying.
		M., 67	Died suddenly fifteen minutes after collapse.
		M., 68	Pains in chest, rested, collapsed and died in street.
		M., 71	Collapsed and died walking along street.
		M., 75	Found dead in afternoon lying across his bed.
		M., 78	Collapsed in street and died.
		M., 83	Found dead at home.

clots were found in branches of the coronary arteries in eight of the twelve, but not in the others, though in several cases the lumen is noted as having been greatly reduced and nearly obliterated in places. Apart from these cases of cardiac infarction, thromboses were found in the coronary vessels in only eight of the remaining fifty-four cases, and in four of these there was fibrosis of the heart muscle. There were fourteen further subjects of fibrosis of the heart muscle with coronary disease. The other thirty-two subjects showed coronary atheroma alone; thirteen of them had hypertrophied hearts and in only four were ante-mortem clots present. Among the younger persons who died from coronary atheroma alone was a man, aged thirty-five years, who collapsed in his bedroom and died fairly quickly; another, aged thirty-seven years, collapsed and died; and a trainer, aged thirty-nine years, who was running to attend an injured football player, collapsed, gasped and died. It will be seen from the above that in many people who die suddenly, all one can find is a varying amount of atheroma in the coronary vessels, reducing the lumen in places. In some of these cases there is evidence, in fibrosis of the heart muscle, that the blood supply had been previously interfered with, but there were no obvious ante-mortem thrombi, at any rate in the larger branches of the coronary vessels. The idea is evidently erroneous that most of these people die from coronary thrombosis. They die from coronary disease; but the exact mechanism of death does not seem to be clearly established. Only six of the 66 were women.

The Prevalence of Fatal Coronary Disease.

In the last 3000 Royal Adelaide Hospital autopsies, it appears probable that 98 of the 98 subjects of cardiac infarction, 59 of the 92 subjects of fibrosis of the heart, and 63 of the 106 subjects of coronary disease of cardiac importance not included in the above—a total of 220 out of the 296 with coronary disease and its effects—died as a result of this. Thus nearly 10% of the 3000 subjects had appreciable coronary disease and about 7% of the 3000 died as a result of this.

Conclusions Based on Adelaide Data.

1. Infarction of the heart muscle from coronary disease has increased from about 11 per thousand between 1920 and 1925 to 45 per thousand between 1943 and 1946. The increase is greater than can be explained by aging of the population, and it cannot be explained by better post-mortem diagnosis. There were 98 cases (3·3%) in the last 3000 autopsies at the Royal Adelaide Hospital.

2. The condition is nearly twice as common in males as in females, but this is offset by the fact that autopsies on males at the Royal Adelaide Hospital are twice as many as autopsies on women. In coroner's cases, in which the subjects were found dead or died suddenly, males much preponderate.

3. In both sexes most deaths occur in the sixties; this incidence is followed by the seventies in males, and then the fifties, with the forties not far behind, and the fifties and seventies in females. One woman was aged twenty-eight years, another was aged thirty-eight years and one man was aged thirty years.

4. As the heart was hypertrophied, sometimes very much so, in 64 out of 109 cases of infarction, it may be inferred that such hypertrophy was a predisposing factor to infarction. This is surprising in view of the necessity for the coronary vessels to increase in size so as to maintain an adequate blood supply to the enlarging muscle. This they do to a considerable extent; and yet, in spite of the larger lumen, thrombosis, or at least obstruction, is more likely to occur to such an extent as to cut off the blood supply to appreciable masses of muscle.

Rupture of, or leakage of blood through, the infarcted area causes sudden or rapid death. As cardiac infarctions are sometimes silent or nearly so (vague pains or symptoms of indigestion *et cetera*), sudden deaths due to this cause are apt to occur in the population at large and so come under the notice of the coroner (eight out of twelve cardiac infarctions) or to occur in mental hospitals (four out of ten). Such sudden deaths are relatively less common in subjects of infarction at rest in bed in general hospitals (12 out of 125).

6. Nearly one-third of subjects of cardiac infarction (32 out of 98) have ante-mortem clots in the chambers of the heart. Such clots were present in the left ventricle in all but one of these, and in this chamber alone in 21 of the 32.

7. An aneurysmal bulge of the degenerated or fibrosed muscle occurred in five males amongst the 147 cases of infarction and the 92 of fibrosis of the heart muscle from coronary disease.

8. There were 92 instances of fibrosis of the heart muscle from coronary disease—that is, 3%—in the last 3000 autopsies at the Royal Adelaide Hospital. Males much preponderated (77 to 15 women).

9. In addition to the cases of infarction and fibrosis of muscle, there were 106 further cases (3·5%) in the last 3000 autopsies at the Royal Adelaide Hospital, in which the coronary atheroma was of such a degree as to embarrass or be likely to embarrass the heart.

10. In these 3000 autopsies it is probable that all the 98 subjects of infarction, 59 of the 92 subjects of fibrosis, and 63 of the 106 subjects of extensive coronary disease, or about 7% of the 3000, died from the effects of their coronary atheroma.

11. In 66 coroner's cases of persons found dead or having died suddenly from coronary disease, 12 subjects had infarcted muscle, 18 had fibrosis of the cardiac muscle, and 36 had as yet neither of these sequelæ. Thromboses were detected in eight of the 12 cases of infarction, in four of the 18 subjects with fibrosed muscle, and in only four of the 36 for whose sudden death no other cause could be found beyond coronary atheroma with more or less constriction of the lumen, though in some instances not much. It cannot be held that all the 32 subjects in which no clot was found had certainly died in some way from their coronary disease, though it seems reasonable to assume that nearly all did so.

SOME ASPECTS OF THE PATHOLOGY OF THERMAL BURNS.[1]

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IN thermal burns, the skin is the only tissue primarily affected, except when the burns are deep and severe or when the respiratory tract is involved by the inhalation of hot air or steam. It must be remembered that the skin is an extensive organ and, considered from the physiological viewpoint, performs several functions in its protection of the body from the oft-changing external environment. The importance of the sensory nerve endings, of the sweat glands and the vast vascular bed with its arterio-venous shunts, and of the rich lymphatic system acting as a protection against bacterial invasion, is obvious.

The Local Lesion.

When the skin is burned, all these functions in the burned area may be upset. Let us first consider the local lesion. This consists of an area of tissue which has been heated sufficiently to cause either coagulation or ultimate dissolution of the cells, so that the tissue will become necrotic. Surrounding this area is a region of varying depth and extent which has been heated to various temperatures, ranging from that of the necrotic area to that of the normal body. It is in this region heated to moderate temperatures that sufficient injury will have been done to damage cell permeability without completely destroying cell structure. Clinically, burns have been classified according to the depth of the injured tissue, but it is obvious that the exact depth of injury is not easily determined, especially at the outset.

The general systemic disturbance which may result from this local lesion has been classified into various stages, all of which may or may not be evident, depending on the extent of the burn, the position of the burn, and the mode of treatment; they are as follows: (i) initial shock, 0-2 hours; (ii) secondary shock, 0-48 hours; (iii) acute toxæmia, 6-100 hours; (iv) septic toxæmia, after about 100 hours; (v) healing. Since infection is now more easily controlled, most deaths are liable to occur from shock or toxæmia, so I shall confine my remarks to the changes which mainly occur in these two stages.

Secondary Shock.

Secondary shock may become evident from the outset and persist for one to three days unless vigorously treated. To understand the ætiology of burn shock we must consider the tissue changes that occur in the local lesion. It is in the partially damaged area that we have to look for the primary cause of fluid loss, for in the tissues completely coagulated by heat there will be no blood flow. For example, if a rabbit's ear is placed in water at 100° C. for half a minute, the whole ear is coagulated and shrivels up with little or no œdema formation; but if the ear is placed in water at 65° C. for the same time, it will ultimately swell enormously.

The production of œdema, then, must be the result of an alteration of the forces governing the fluid exchange through the damaged capillary membrane where the tissues are not completely destroyed. In the normal capillary, the fluid exchange through the membrane is the result of the balance of three forces: (i) hydrostatic pressure in the capillaries, (ii) effective osmotic pressure of the plasma proteins and (iii) tissue tension. The skin does not normally become œdematous because the capillary membrane is relatively impermeable to the large protein molecules. Some protein does, however, escape and is returned to the blood-stream by the lymphatics.

Now let us see how the permeability of the capillary membrane alters when the skin is heated. This may be observed by cannulating the lymph channel in the foreleg of a dog and estimating the flow and protein content of the lymph coming from the paw when this paw is immersed in water at different temperatures (Courtice, 1946). Warming the paw from room temperature greatly increases the blood flow. The arterioles are dilated, and so the capillary hydrostatic pressure increases with resultant increase in capillary filtrate and a slight increase in lymph flow. The protein content of lymph, however, falls, because there is an increase in the amount of capillary filtrate, containing a small amount of protein, and a decrease in the amount of non-protein fluid reabsorbed by the blood capillaries, which results in a fall in the protein concentration of the tissue fluid. When the temperature is increased to 55° C., however, the lymph flow rises rapidly, as does the protein concentration in the lymph. The lymph protein level reaches a value approximately equal to that in the plasma. This can mean only that the permeability of the capillary membrane to the plasma proteins is increased, so that the osmotic effect of the plasma proteins is cancelled out, the hydrostatic pressure exerts its full effect, and œdema of the damaged area rapidly results.

If the damaged area is large enough, this rapid outflow of fluid will be considerable and will upset the fluid balance of the whole organism. There is normally a delicate balance between the three litres of plasma on one side of the body's capillary membranes and about 12 litres of tissue fluid on the other side. The immediate effect of the plasma loss into the damaged tissues will be to decrease the plasma volume. As far as dynamics of the circulation are concerned, this is the primary change in burn shock. The decreased plasma volume will lead to a decreased venous return, which in turn will lessen the cardiac output, so tending to cause a fall in blood pressure. A fall in blood pressure will immediately bring into play the carotid sinus and aortic arch reflexes, which reflexly cause vasoconstriction of the vessels of the undamaged tissues, except the brain, where the vasoconstrictor nerves have little effect on the calibre of the blood vessels. Should the plasma volume continue to fall, a time will come when this vasoconstrictive compensatory mechanism will fail; the volume of blood becomes too small for the volume of the vascular bed and irreversible peripheral circulatory failure occurs.

Another, but slower, compensatory mechanism is also brought into play to lessen the fall in plasma volume—the reabsorption of fluid from the non-injured area. Thus the actual fall in plasma volume will depend upon the imbalance of two processes, the rate of the passage of plasma into the burned area and the rate of reabsorption of fluid from the undamaged tissues.

The changes in the blood will primarily depend upon these two rates. At first the outpouring of plasma is usually greater than the reabsorption of fluid, so hæmoconcentration results. This is evident from the rise in the hæmatocrit reading, or the hæmoglobin content of the blood. This hæmoconcentration is one of the most outstanding features of a severe burn, and its measurement is the most useful guide in the early therapy of burn shock. The hæmatocrit reading is much more useful than the blood pressure reading, since vasoconstriction will maintain the blood pressure within normal limits even though the loss of plasma is considerable. In the early stage of burns, therefore, the hæmoglobin content or hæmatocrit reading should be estimated frequently to be used as a guide for fluid therapy. Another effect of the alteration of fluid balance is the fall in plasma protein concentration. This is due primarily to the loss of fluid rich in protein and the reabsorption of a protein-free fluid.

All experimental evidence points to the local loss of fluid as the fundamental cause of secondary shock in burns. The literature on this subject is considerable (Harkins, 1942, 1945), but the following evidence will suffice to support this view.

1. Is the loss of plasma in the burned area sufficient to cause the circulatory failure in shock? Harkins (1935) has estimated that if one side of the trunk of a dog is burned, the plasma loss may be 3·5% of the body weight, which is more than half the total plasma volume. I have shown (Courtice, 1946) that if two hind legs of a rabbit are scalded just above the knee, the œdema fluid may be equal to or greater than the total plasma volume. Cameron, Allen,

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 25, 1949.

Coles and Rutland (1945) have similarly shown in goats a considerable loss of fluid in the burned area.

2. If the local loss of fluid is decreased or prevented after a burn, can the signs of shock be prevented? Local fluid loss can be greatly reduced in burns, either by reducing the capillary hydrostatic pressure or by increasing the local tissue tension, since these are the only two forces regulating fluid exchange across the capillary membrane. I have decreased the hydrostatic pressure by keeping the burned legs of rabbits cold. By this procedure, the arterioles are constricted and the blood flow through the affected area is decreased with a resultant lessening in the rate of œdema formation. With a decreased rate of œdema formation, hæmoconcentration and the fall in blood pressure are considerably decreased. Similarly, if the tissue tension is raised by the application of pressure bandages, the œdema formation and the circulatory changes in shock can be greatly lessened (Courtice, 1946; Cameron *et alii*, 1945).

3. Is there a generalized loss of fluid in burn shock? Fine and Seligman (1943), using plasma containing a radioactive element in the protein molecule, found that there was no abnormal leakage of protein through non-burned tissues except probably in the very late stages near death. Glenn, Muus and Drinker (1943) and Cameron, Courtice and Short (1947) have shown by cannulating lymphatics that the permeability of the capillary membranes to proteins was not increased elsewhere than in the burned area.

It seems, therefore, that the local loss of plasma into the damaged area is the primary cause of burn shock. Shorr, Zweifach and Furchgott (1945), however, think that in recent years the pendulum has swung too far towards the fluid-loss theory of the causation of shock. They have made detailed observations on the hepato-renal factors in shock. Fluid loss initially is still recognized as the primary factor. In the early stages this produces renal ischæmia, and anaerobic processes in the kidney rapidly form and release "V.E.M." (vasoexcitatory material), which is responsible, in part at least, for the compensatory vasoconstriction. During this hyperreactive stage the liver does not form "V.D.M." (vasodepressor material), but retains its capacity to destroy any "V.D.M." which is slowly produced in the skeletal muscles.

The stage of hyperreactivity passes gradually into a hyporeactive stage, in which amounts of "V.D.M." appear in the blood-stream and the terminal arterioles become quiet and unreactive. Blood is directed to capillary venules and stagnates. Hepatic anoxia starts anaerobic metabolism in the liver with rapid production of "V.D.M." and progressive deterioration and failure of the "V.D.M."-inactivating mechanisms. At the same time the kidney loses its capacity to form "V.E.M.". Thus at this stage transfusions may be unsuccessful since the transfused fluid or blood continues to accumulate and stagnate in the venules.

Mazur and Shorr (1948) have recently identified "V.D.M." with ferritin, an iron-protein, and its iron-free component, apoferritin. Their work indicates that in addition to iron transport and storage, the ferritin-apoferritin system plays an important role in the regulation of the peripheral circulation.

This work on the hepato-renal mechanisms in shock is still in the experimental stage, but it suggests that the condition of shock in burns must be tackled vigorously in its early stages, in an endeavour to prevent the later hyporeactive state of the blood vessels due to the anaerobic formation of "V.D.M." in the liver.

Toxæmia.

The questions whether toxins are formed in the locally burned tissues, and whether if formed they play an important role in the production of morbidity and mortality following burns, are the subject of much controversy and speculation. The literature is considerable and has been reviewed by Harkins (1942, 1945). A clear picture of burn toxæmia has been clouded from the beginning by other factors. In the first place, the part played by fluid loss in shock was not properly appreciated until comparatively recent times, with the result that early workers were seeking a toxin produced in the burned tissues to explain the shock syndrome. Many such toxins were postulated by experimentalists. Since it has been shown that shock is mainly due to fluid loss and now can generally be combated by vigorous therapy, a toxin has been sought to explain symptoms which may arise later when shock no longer exists. But this period of so-called toxæmia has in the last two decades been complicated by what appears to be the toxic action of tannic acid causing a specific central necrosis of the liver. In addition, infection must have clouded the picture of toxæmia, especially before the introduction of antibiotics. So the whole literature on the question of toxæmia in burns is most difficult to assess.

Histamine has figured largely in the controversy. It appears that there may be a rise in the histamine content of the blood in burned patients (Barsoum and Gaddum, 1935), but these authors found no evidence of any correlation between the blood histamine level and the clinical condition of the patient. Rose and Browne (1942) noted an early rise in blood histamine level followed by a decrease at the onset of toxæmia and œdema. This was followed by a rise with the clinical improvement of the patient. Kellaway and Rawlinson (1944) found that histamine was liberated from the hind legs of guinea-pigs when these were perfused at temperatures from between 45° C. and 50° C. It seems, therefore, from this and other experimental evidence that histamine may be liberated from the burned tissue, but what part it plays in burn toxæmia is not clear.

Leach, Peters and Rossiter (1943) have postulated that in the moderately heated area sufficient injury to the tissues may have been done to damage cell permeability without altering the nature of some of the enzymes. These enzymes may then diffuse out and prove to be toxic in parts of the body to which they are foreign. Kellaway and Rawlinson (1944) showed that in perfused limbs alkaline phosphatase, lipase and proteolytic enzymes were set free at temperatures above 41° C. They conclude that the demonstration that lipase and proteolytic enzymes are set free gives colour to the possibility that toxic products may be formed by enzyme activity in the tissue spaces.

Clinical evidence indicates that certain patients with burns die after the period of secondary shock and before the onset of infection. Histological changes in this toxæmic stage are largely centred in the liver. Liver changes have been described by many authors, but the work of Wilson and his colleagues (1938) added renewed interest to this aspect of burn pathology. They describe 33 fatal cases, in which the most striking feature of death after the first twenty-four hours was central necrosis of the liver. A little later, during the war in the Middle East, Wilson suggested that tannic acid might be absorbed and cause this liver damage. Since then, much evidence has accumulated to indicate that tannic acid is probably the chief cause of focal central necrosis of the liver in burns.

In animals it has been shown that tannic acid injected subcutaneously can produce liver necrosis (Cameron, Milton and Allen, 1943; Clark and Rossiter, 1943). Cameron *et alii* also showed that in goats in which an experimental burn was treated with tannic acid, there was a measurable quantity of tannic acid in the blood and liver damage was evident. They concluded from this study on experimental animals that there seems little doubt that even with not very extensive skin burns, the application of tannic acid may be followed by its appearance and persistence in the blood and the development of liver damage. From the clinical aspect, several papers have also appeared which indicate that tannic acid is the cause of liver necrosis (Belt, 1939; Erb, Morgan and Farmer, 1943; Wells, Humphrey and Coll, 1942).

It seems evident that in extensive burns, tannic acid may be absorbed and cause central necrosis of the liver, so the whole question of burn toxæmia during the past twenty four years since the introduction of tannic acid treatment is complicated by this finding. Burn toxæmia, therefore, remains a controversial question, and much work, both experimental and clinical, is still needed to elucidate this problem.

Nitrogen Metabolism in Burns.

Another subject important in the treatment of burns is nitrogen metabolism. In burns, as in many other injuries or infections, there is often a negative nitrogen balance, that is, the nitrogen output is greater than the nitrogen intake. This can mean only the breakdown of body proteins with wasting.

The degree and importance of nitrogen loss in burns seem to vary with different authors. Cope, Nathanson, Rourke and Wilson (1943) carried out a detailed nitrogen investigation in a group of patients from the Cocoanut Grove disaster. They showed that there was always a negative nitrogen balance during the first week, but during this time the caloric intake was inadequate. Later nitrogen equilibrium was easily established when caloric and nitrogen intakes were increased. Cope was surprised how easily this could be done and attributed it to the lack of infection. It is well known that infection will greatly aggravate the negative nitrogen balance and make it almost impossible to maintain nitrogen equilibrium. Hirshfeld, Abbott, Pilling, Heller, Meyer, Williams, Richards and Obi (1945) have also made a detailed study of the nitrogen balance of a group of burned patients. They showed that practically all their patients were in a negative balance and that it was possible to decrease this and diminish loss of weight by feeding diets of high protein and caloric content. But they found that in the first few days after injury, neither these diets nor intravenous injections of hydrolysates were well tolerated.

The question of the cause of the negative nitrogen balance is not easy to answer. In the early stages three factors probably play a part: (i) loss of plasma protein into and from the burned area with the result that the body tissues must make good this loss; (ii) lack of nitrogen intake because the patient has anorexia and probably vomiting; (iii) dehydration which, however caused, will produce a breakdown of body protein.

The nitrogen loss, however, may persist with severe wasting. In the later stages infection may aggravate the loss, but apart from infection it appears that other factors probably come into play. Croft and Peters (1945) postulated that specific amino-acids, such as methionine, were necessary to repair skin damage, and to provide sufficient of these, much body protein must be broken down. At first it seemed that feeding extra methionine in controlled experiments on rats had a beneficial effect, but later work disproved this. Other authors have tried to correlate the nitrogen loss with damage to the adrenal glands. Cope *et alii* showed in their patients that the 17-ketosteroid excretion was elevated during a period corresponding to the negative nitrogen balance, but they could come to no definite conclusion regarding any relation between 17-ketosteroid excretion and nitrogen metabolism.

Anæmia in Burns.

In severe burns, hæmoconcentration is often followed by progressive anæmia, which remains until the granulating surfaces are healed. The anæmia is probably due not to one but to several causes:

1. Destruction of a significant number of erythrocytes by heat, depending on the temperature attained by the blood, the duration of heating and the volume of blood subjected to these conditions. If red cells are heated *in vitro* to approximately 53° C., they become more fragile and are prone to fragmentation. In patients with severe burns, hæmoglobinæmia and hæmoglobinuria with hæmoglobin casts in the kidney have been noted.

2. Loss of blood from the burned area and later from the granulating surface.

3. Dehydration. Most types of severe dehydration are usually followed by anæmia, irrespective of the cause of the dehydration.

4. Malnutrition with hypoproteinæmia and later sepsis, which may cause continued destruction of erythrocytes or slow down their formation.

Conclusion.

In this brief lecture, I have tried to give an outline of the more obvious pathological changes which require vigorous treatment from the outset. Time does not permit me to deal with renal changes, the possible significance of the suprarenal glands or other less common pathological disorders which may be observed in burns. The questions of infection and of healing will be dealt with by Dr. Rose.

It is evident that the damage of a fairly large area of the protective covering of the body may set in motion a series of pathological changes which will rapidly lead to death if untreated. It is equally evident that the pathological laboratory must play an important part in assessing the progress of a patient inflicted with serious burns, and it seems that in such cases full use should be made of the laboratory in guiding the clinician who is treating the patient.

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CONTROLS IN THE TREATMENT OF BURNS.[1]

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PROPHYLAXIS is the keynote in the ideal management of burns and scalds. Shock, starvation, infection, deformities and contractures are avoidable complications, to be prevented whenever possible rather than treated. Clinical, bacteriological, hæmatological and biochemical controls are a necessary aid in this achievement.

Civilian burns, as opposed to war and air-raid burns, are mostly of minor extent, and only 10% cause any trouble in their treatment (Lam, 1948). Only 51 (31%) of the 163 patients admitted to the Royal North Shore Hospital in the past ten years had more than 10% of skin burnt. Only six had any considerable full-thickness loss of skin.

Prevention and Treatment of Shock.

Burn shock, like shock in other injuries, consists of two phases, the neurogenic and the oligæmic.

Neurogenic Shock.

Some authorities deny that there is a neurogenic shock, pointing out that fluid loss and concentration of red corpuscles are found immediately a burn is incurred. However, there can be no doubt that neurogenic shock is present to a considerable extent in patients suffering burns due to explosives, gas-oven explosions and air-raids. The last-mentioned patients are often in a state of shocked terror, as they know that they still run a risk of further bombing. Neurogenic shock is also well seen in burns of the face, hands and perineum. It is pronounced in children and in the elderly, even with small burns. The intravenous injection of morphine is the treatment of choice in these cases.

Pain is rarely a pronounced feature of burns, and if it is, it quickly responds to the intravenous injection of morphine. The intravenous administration of procaine is now being tried (Abbott and Hirshfeld, 1947; Wulf and Grunert, 1948; Barbour and Tovell, 1948; Schalit, 1949); but it is easier to give a quick injection of morphine than saline with procaine intravenously. Dr. C. N. Paton, honorary anæsthetist to the Royal North Shore Hospital, tells me that he used this drug after thoracoplasty, but the results were too inconstant, and the dosage causing loss of pain and that causing convulsions were too close for safety.

Oligæmic Shock.

Fluid loss in burns of slight extent can be made good orally unless vomiting occurs. Adult patients with burns involving 10% or more of the body surface and children with burns involving 5% or more[2] will require intravenous therapy to prevent or treat oligæmic shock. In this regard, it is interesting to note that at the Royal North Shore Hospital only 15 of the 51 patients with more than 10% of surface area burnt, appeared to require intravenous therapy. Many of these cases, however, occurred in the days before the value of resuscitation was fully appreciated.

The oligæmic phase of shock due to the "white hæmorrhage" of plasma onto the surface of the wound and into the injured tissues commences as soon as the burn is suffered and before hæmatological investigation reveals any hæmoconcentration (Rosenqvist, 1947). Thus it is essential to prevent the appearance of the signs of oligæmic shock by the immediate use of parenteral therapy in all such cases, even if the patient looks well and has a normal blood pressure and pulse rate and a normal hæmoglobin or hæmatocrit value. In cases of doubt the patient is always regarded as seriously ill. Of course, if frank oligæmic shock is present, then intravenous therapy must be intensified. This period of fluid loss from the circulation lasts for forty-eight hours or more, so that parenteral therapy will be necessary during this time even if the patient can take fluids by mouth.

There are two objectives in the fluid therapy of burns. The first is the rapid parenteral treatment of acute fluid deficiency in the first forty-eight hours whilst fluid is being lost from the circulation into the injured tissues. The second is the maintenance of normal daily fluid needs, which usually can be achieved orally after forty-eight hours unless toxæmia or infection arises.

Until recently, plasma was used in the treatment of oligæmic shock in burns, but it has now been replaced by whole blood and electrolyte solutions (physiological saline). Plasma, in England at least, is under a cloud owing to the frequency of homologous serum jaundice following its use.

The rationale of the use of whole blood is based on the fact that there occur a destruction of red cells and spherocytosis (Shen, Ham and Fleming, 1943), and also a reduction of the total red cell mass following a burn. As a consequence, whole blood does not increase hæmoconcentration (Moore, Peacock, Blakeley and Cope, 1946). Also it has been found experimentally (Moyer, Coller, Iob, Vaughan and Marty, 1944; Abbott, Meyer, Hirshfeld and Griffen, 1945) and clinically (Abbott, Pilling, Griffen, Hirshfeld and Meyer, 1945; Evans and Bigger, 1945; Lange, Campbell and Coller, 1946; McDonald, Codman and Scudder, 1946) that if whole blood is used, the later anæmia of severe burns is not so pronounced. Whole blood also prevents liver anoxia and so lessens later hypoproteinæmia. Plasma has not the same effect.

Electrolyte solutions are necessary too, because sodium is lost as quickly as plasma (Moore, Peacock, Blakeley and Cope, 1946).

Immediately it is decided that a patient requires fluids parenterally, an infusion of a litre of plasma (which does not require typing) is commenced. Whilst this is being given, the patient's blood is typed and cross-typed against suitable donor blood. Rh grouping is mandatory as numerous transfusions may be necessary. Hæmoglobin or hæmatocrit estimations are performed on the capillary blood (this is as efficient as using venous blood—Owens, 1947). These first readings may not give a true picture of the amount of oligæmia in early cases, but they act as a basic reading for later comparison. They are repeated at intervals of three hours to help control the amount of blood given. Blood and glucose-saline solution are then administered in order to keep the hæmoglobin level normal. The saline also helps to keep the body well hydrated. This will be reflected in the state of the tongue and the flow of urine, an excretion of 100 millilitres per hour being desired. Oliguria may be present in the first few hours, as the first fluid goes to make up tissue fluid loss. The salt output is measured by Fantu's test (Hutchinson and Hunter, 1949), a minimum excretion of three grammes of sodium chloride per litre of urine per day being aimed at. This simple test is as efficient as the more complicated blood analyses for sodium and chloride. If the fluid intake is sufficient, any acidosis will automatically right itself and require no special treatment (Marriott, 1947).

As much as 15 litres of fluid may be required in the first twenty-four hours in very severe cases. It may be necessary to commence by using two venous infusions at once, so as to give the first litre of fluid in twenty minutes, the next in forty minutes, and the third in one hour, and so on for forty-eight hours in lessening amounts.

In addition to parenteral therapy, if the patient is not vomiting (in severe shock vomiting may be such as to require continuous stomach aspiration), one gives fluids by mouth, such as water, fruit juices, tea or milk. Two litres per day in burns involving under 10% of the body surface, or three to eight litres in more severe cases, may be given on the first day, and so on, according to the patient's ability to take and retain the fluid.

In order to prevent the administration of excessive fluids, a fluid record is kept, the chest is watched and the urinary

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 25, 1949.

[2] Harkins (1945) points out that the face and neck, the dorsal or ventral surface of the chest or the abdomen, one upper extremity, or the dorsal or ventral surfaces of one lower extremity, are all approximately 10% of surface area.

output is measured. Plasma proteins may be estimated to prevent the level from falling below five grammes *per centum*.

By these means a normal blood volume with an adequate hæmoglobin concentration is obtained, dehydration, acidosis and salt depletion are corrected, and an adequate urinary evacuation is maintained.

Prevention and Treatment of Protein Loss.

Forty-eight hours after a burn is sustained, a microcytic anæmia may occur, its severity depending on the extent of the injury. This anæmia is due to a combination of causes. Blood as well as serum may be lost into the burnt area, especially in full thickness burns. More important than this, however, is a deficiency of hæmatopoiesis due to the loss of tissue protein (Braithwaite and Moore, 1948; Lyons and Mayerson, 1947). In fact, the blood count gives as true a picture of protein loss as the estimation of the level of plasma protein.

The cause of this protein deficiency is manifold. There is the usual non-specific nitrogen loss which occurs after any injury (Braasch, 1949). There is loss of protein onto the burnt surface and into the surrounding tissues. There is lessened protein intake due to anorexia and sometimes vomiting. In addition, severely burned patients suffer hepatic damage as revealed by liver function tests (Boyce, 1942). This is a fatty degeneration of the liver cells due to the anoxia of severe shock or to later protein deficiency causing a lack of lipotrophic substances. It differs from the liver damage caused by tannic acid, which is a centrilobular necrosis. This liver damage affects the formation of plasma albumin, with which the tissue proteins are in a state of dynamic equilibrium.

Cuthbertson (1945) has shown that a severely burnt patient may lose four pounds of protein in ten days, so that such a patient needs nutritional resuscitation as well as shock resuscitation.

Loss of protein causes lack of healing of the burnt area, lack of healing of the donor graft area, and failure of grafts to take. This causes a vicious cycle because it allows further protein to escape. Thus early epithelialization stops protein loss.

In any injured patient there are priorities in protein repair: firstly the blood, then wounds, then tissue protein and plasma globulin, and lastly plasma albumin. Consequently, repeated blood transfusions will counteract the anæmia, at least temporarily, whilst other methods of protein replacement can go straight to the wounds and lower priorities of tissue repair. The amount of blood to be given is regulated by the results of frequent blood counts.

The main method of protein replacement is by oral feeding. Patient with burns covering 20% of surface area require 300 to 400 grammes of protein per day with a 5000 Calorie diet (25% of these Calories must be derived from protein and less than 15% from fat—Levenson, Green and Lund, 1946). In fact, five times the normal daily amount of protein intake may be needed. The dietitian of the Royal North Shore Hospital has prepared diets for me each yielding 3000 Calories and containing 200 grammes of protein to give in the various stages of treatment. A semi-solid diet (see Appendix A) is used as soon as the patient can take it, usually from the third day on. He then graduates to a more solid diet (Appendix B). If much vomiting occurs, gavage may be successful and a liquid diet is given (Appendix C).

It may be noted that dried skimmed milk is used in these diets and not aminoacid mixtures, such as "Essenamine", which are not easy to take.

In addition vitamins are required, the daily amount being one to two grammes of ascorbic acid, and 50 milligrammes each of thiamine, riboflavin and nicotinamide.

If the patient is so ill that oral feedings cause vomiting and gavage is unsuccessful, one may need to resort to intravenous protein therapy. A protein hydrolysate, such as "Parenamine", (Elman, 1947) may be used and as much as 225 grammes per day in a 10% solution has been given (Levenson, Green and Lund, 1946). However, much of the amino acids of the solutions are wasted by rapid excretion of their nitrogen by the kidneys before they have a chance of reaching the liver (Magee, 1948). It has been shown that serum (Isbister, 1948) and whole blood (Hoffman, 1947) are far more efficient in yielding usable nitrogen. In addition, these aminoacid solutions tend to cause clotting of veins, and veins are very precious in these cases.

In this phase of protein loss one needs frequent blood counts and possibly plasma protein estimations, but most important of all are frequent clinical appreciations of the patient so that any necessary grafting can be performed as soon as possible.

Prevention and Treatment of Infection.

The avoidance of infection in burned patients is the central problem of treatment after management of the period of shock. Infection increases the tissue damage, delays healing, prolongs stay in hospital and increases the mortality rate.

In hospital, we must make a fourfold attack on infection. Firstly, no pathogens must be allowed to reach the patient at dressings; secondly, none must be allowed to reach the patient between dressings; thirdly, any reservoir of pathogens must be eliminated; fourthly, raw areas must be epithelialized quickly—if not naturally, then by grafting.

Colebrook and his associates (1947, 1948), in their work at the burns unit of the Birmingham Accident Hospital, have placed the prevention and treatment of infection of burns on a firm basis.

All burns when incurred are sterile owing to the heat. If the patients come immediately to hospital, the majority are still uninfected. It is in hospital that infection occurs unless the greatest of care is taken. Burns are not only infectable but also infective, so that the management of burns infection is the management of hospital infection.

The common pathogens involved are the β-hæmolytic streptococcus (Group A), the hæmolytic *Staphylococcus aureus*, and later *Proteus vulgaris*, *Bacterium coli* and *Pseudomonas pyocyanea*. These Gram-negative organisms may not be so lethal as the cocci, but it was found in war wounds that these organisms caused much suppuration and prolonged the time of healing and delayed wound covering. They are, in fact, far from being the negligible factor that was once thought.

These pathogens come from numerous sources. The respiratory tracts of the attendants and the patient himself may furnish the streptococcus. The nose may harbour the staphylococcus, as also may furuncles and skin infections of the patient. The patient's alimentary canal may furnish *Bacterium coli* and *Proteus vulgaris* especially in buttock wounds. The attendants' hands, the patient's clothes, the bedclothes (especially blankets), and improper methods of wound dressing in a general ward with infected surgical patients, may cause contact infection. Air-borne infection may be caused in a general surgical ward when septic conditions are present. There are numerous pathogens in the dust of the ward, either floating in the air or resting on the bedclothes or in the dust on the floor. Colebrook and his associates (1948) showed that burns dressed in such a ward have a higher percentage of infection than those dressed in a special room. They therefore abandoned ward dressings and virtually eliminated both air-borne and contact infections by carrying out all dressings under aseptic operating room conditions in a special room set aside for this purpose. This was equipped with the use of filtered air (Bourdillon, Lidwell and Lovelock, 1949), so that air-borne infection at the time of dressing was eliminated. Proper masking of the patient and attendants and the use of penicillin have eliminated streptococcal infection in Colebrook's unit.

The real danger of infection then lies in the period between dressings if the patient is nursed in a general ward, especially if infected patients are present. The organisms in the dust of such a ward can infect any burn whose dressings have slipped and exposed some of the raw area. Again dressings soaked in serum may become contaminated from without (Colebrook and Hood, 1948). An infected burn is a danger to other patients in a general ward, as it is a reservoir of pathogens. To eliminate this,

wards must be air-conditioned, blankets must be oiled and autoclaved frequently (five pounds' pressure for twenty minutes) and the floors must be vacuum cleaned rather than swept and treated with spindle oil.

A burns unit run on the lines suggested by Colebrook with single rooms or cubicles and with its own air-conditioned operating theatre will solve many of the problems of infection. Certainly Colebrook's results are impressive. He has eliminated β-hæmolytic *Streptococcus hæmolyticus* (Group A) for the past three years. But *Pseudomonas pyocyanea* and the staphylococci are still the bugbear in his wards, just as the staphylococcus is in all hospitals. In fact, 60% of his patients become contaminated with staphylococci in hospital at one time or another. Many of these staphylococci are unfortunately penicillin-resistant. Some may respond to streptomycin and others to aureomycin, which has been shown recently to be effective against Gram-positive and Gram-negative organisms including staphylococci (Yeager, Ingram and Holbrook, 1949; Nichols and Needham, 1949), as well as against viruses.

Of course, too, infection should be prevented before the burnt patient reaches hospital. This requires the education of the public to leave such a patient alone as far as possible and not to cover the burn with doubtfully clean blankets.

Consequently, this phase of burn treatment requires many controls, administrative and otherwise. It entails a multitude of bacteriological controls, swabbings, cultures and sensitivity tests to penicillin and streptomycin.

Failing the attainment of a burns unit, much can still be done to combat the risk of infection by nursing a burnt patient in a single room and dressing his wound in an operating theatre.

Treatment of the Burnt Area.

As soon as the patient arrives in hospital, a prophylactic parenteral course of penicillin should be commenced; 100,000 units of ordinary penicillin, given every eight hours, or 300,000 units of "Procillin" given once a day, are satisfactory, as we know now that penicillin does not require a constant high blood level for its effect (Tillett, Cambier and McCormack, 1944; Jametz, 1946; Zubrod, 1947; Marshall, 1948; Sanders and Lockwood, 1949).

The local treatment of the non-infected burn aims at keeping it from becoming infected. The dressing should not increase epithelial loss. It should allow drainage and exert moderate pressure. It cannot prevent serum from escaping as its original exponents intended.

Until recently, after shock was treated, the burnt area was cleansed with white soap and water or a solution of "Lux". All dead skin was removed and blisters were snipped. The raw area was then rinsed with saline and sprayed with penicillin-sulphanilamide powder. *Tulle gras* was applied and affixed with gauze bandages moistened in saline. This was then covered with cotton wool and the whole bandaged with a *crêpe* bandage.

Under this régime, 16 "clean" burn patients were treated without penicillin given parenterally, and one became infected (with *Staphylococcus aureus*); 42 "clean" burn patients were similarly treated, but with penicillin given parenterally, and six became infected (two with both streptococci and staphylococci, one with staphylococci and *Proteus vulgaris*, one with staphylococci alone, and two with *Proteus vulgaris* alone).

However, in 1943, Meleney showed that soap washing of a burn did not reduce bacterial contamination, so that recently I have followed the lead of others (Levenson, Green and Lund, 1946; Elman, Merry, Regnesse and Tisdale, 1946; Lam, 1949) in using a much simpler method of local treatment. Whenever possible the patient is admitted to a well ventilated single room. Shock is treated first, and whilst this is being done, the burnt area is covered with *tulle gras* and pressure bandages with cotton wool are applied. This is performed under aseptic operating room conditions. No washing of the area and no snipping of blisters is performed. Only obviously dead skin is picked off. This single dressing requires no further anæsthetic than the morphine given to treat the shock. If the shock is so severe that not even this can be done, then the burns are covered merely with sterile sheeting to prevent the lodgement of pathogenic organisms and the pressure bandages are applied later.

I do not use penicillin-sulphanilamide powder. I had some rather anxious moments from absorption of the sulphanilamide in tropical areas, and so do not use it, at least for uninjected burns. It has a toxic effect on the lower nephron, which is also found to be affected at post-mortem examinations on subjects of severe burns.

The dressing is undisturbed for seven to ten days then changed in the operating theatre. Then we can see if the burn is epithelialized or covered with flat pink granulations ready for grafting, or, if it is a full thickness burn, whether the slough is ready to be excised.

If it is seen to be an obvious full thickness burn at the original dressing, it is treated with wet dressings from the start (Clarkson and Lawrie, 1946)—tubes are put in the cotton wool and saline is run in slowly by a continuous drip apparatus so as to convert the dry gangrene to a wet gangrene, to permit its earlier excision.

If the affected area becomes infected or is already infected when the patient is first examined, it is swabbed and organisms are identified. The parenteral administration of penicillin is continued, and if Gram-negative invaders are present then streptomycin is given parenterally, one gramme per day. Locally, after any sloughs have been removed and the wound has been cleansed with saline, one may apply penicillin cream or penicillin-sulphanilamide powder, which quickly obliterates any streptococcal infection. If Gram-negative invaders are present, "Propamidine" ointment is very useful. Such patients will require much more frequent dressings than those with uninfected burns, and all must be carried out under operating room conditions with cyclopropane anæsthesia if morphine is not sufficient.

Baths were once popular, and may still be useful if one can be sure that the bath can be efficiently rendered sterile between patients to eliminate contact infection.

At each dressing the wound is swabbed and cultures are attempted, especially for streptococci whose presence alone will stop a graft from taking.

In passing, I may add that I am sure that tannic acid and other coagulation methods of treatment were given up, not so much for the fear of liver necrosis which is now known not to be the cause of burn deaths (Beuman, 1946; Edwards, 1948), nor for the fear of local injury (Saltonstall, 1945), but because of the risk of infection under the coagulum. At the Royal North Shore Hospital, in tanning days, 42 of 93 "clean" burns became infected. I do not know the organisms involved, as bacteriological control in those days was not so strict as it is now; but the notes of "pus under tan", "area around burn red and lymphangiitis present", leave no room for doubt.

Prevention of Contracture and Deformity.

The modern classification of the depth of burns is that of partial skin loss and complete skin loss. Burns in the former category may be subdivided into two types. There are, first, the superficial partial thickness skin loss, recovery from which is followed by the regeneration of normal skin in ten days, and secondly, the deep partial thickness skin loss, in which the burn affects the corium, but the epithelial elements of hair follicles and sweat glands are still present. New skin regenerates from these scattered islets of epithelium, but it is thin and slowly growing. In full thickness skin loss, an eschar is formed and separates later as a slough. This may take seven or more weeks if left alone. Here epithelialization can occur only from the skin edges.

The object in the treatment of all burns is to achieve early epithelialization, naturally in superficial burns or by graft in deeper burns.

Though it may be suspected from the agent causing the burn—for instance, boiling fat will cause a deeper burn than boiling water—it is usually impossible to tell immediately the depth of a burn. Certainly, a superficial blister burn has a red base, in the deeper burn pinpoint

red papillæ are present, and in the full thickness burn a white eschar is seen. However, we cannot tell how deeply the damage goes. The burnt area has four zones of depth—namely, the layer destroyed, then the layer injured which will later die, then the layer injured which will recover, and lastly the subjacent uninjured layer (Meleney, 1949). As a consequence, while it is correct to compare a full thickness burn to dry gangrene, it is incorrect to compare a full thickness burn to a clean surgical wound. Again, though a granulating area after a full thickness burn may look like that after any wound, there is this difference: in the full thickness burn interstitial fibrosis is much more extensive, with deeper penetration, and its formation continues longer. This is due to heat penetration, which sets up a widespread inflammatory reaction (Matthews, 1943); this, if allowed to continue, will cause fibrous contracture.

Early epithelial covering, by preventing infection and further fibrosis, helps to control this. But on account of the unknown depth at first, immediate skin grafting is not always practicable (Lam, 1949).

Fortunately, in civil life full thickness burns are uncommon compared to the blister burns. Of the 163 burns investigated at the Royal North Shore Hospital, only six appeared to be full thickness burns and required grafting. From the long history of some of the earlier cases, however, it would appear that more could have done with grafting.

The ideal time for review of the burn is seven to ten days after the initial dressing. The dressings are removed in the operating theatre as before. The burn will be completely epithelialized if it is a superficial blister burn. In deep partial thickness burns, flat pink granulation tissue will be present with islets of epithelium growing in scattered areas. If left alone this will take a long time to heal, and it should be grafted. This is done by sheet grafting by means of a Padgett's dermatome or a Blair knife or a Humby knife. The split skin graft, 10 to 14 hundredths of an inch, is taken directly from the donor to the recipient area without being placed in saline. Both areas are then treated similarly by penicillin-sulphanilamide powder (omitted by some) and the application of a *tulle gras* dressing reinforced by cotton wool and *crêpe* bandage to exert even pressure. If there is any question of infection of the area to be grafted it is redressed without grafting. Material for culture is taken, and if streptococci are present the burn is treated with penicillin cream or penicillin-sulphanilamide powder and no grafting is attempted until they are eliminated. Otherwise, the grafts will not take.

If the burns are very large, grafting may have to be done in sessions. For small children with extensive burns, it may be rarely necessary to use homografts from the mother to tide the child over temporarily.

This early grafting stops protein loss, infection, fibrosis and joint contracture. It allows early painless movements of the joints. In certain cases the split-skin grafts may have to be later replaced by means of more complicated procedures such as rotation and sliding flaps and pedicle flaps.

In eschar burns, the slough is removed surgically about seven to ten days after the burn has been sustained. If the area beneath is clean it can be grafted immediately; if not, it is prepared in the usual way for a few days (Cope, Langohr, Moore and Webster, 1947; McCorkle and Silvani, 1945). The graft is inspected a week later and it should have taken.

In certain chronic cases, in which grafting will not take, it has been shown that dl-methionine is a specific in epithelial healing. Four grammes are given three times a day (Localio, Gillette and Hinton, 1949).

During the illness, the physiotherapist is called in to help keep joints moving. Later the services of the occupational therapist are necessary.

Burn Toxæmia.

The causation of toxæmia occurring in severe burns is still uncertain. Whilst it is made worse in its early stages by the oligæmia of severe shock, or in its later stages by the toxins of infection, it can still occur in burns the shock from which has been treated or even prevented by adequate fluid therapy, and which remain uninfected until death (Aird, 1949). Consequently, burn toxæmia is due to more than fluid loss, just as oligæmic shock is due to more than fluid loss (Shorr, Zweifach and Furchgott, 1948). Clinically, a case reported by Newson and Armstrong (1945) is of interest, in that it seems to show that a toxin is present in burn toxæmia. A soldier sustained petrol burns involving 50% of his body surface; bilateral toxic retinitis developed on the second day despite adequate fluid replacement therapy.

Consequently, at present, until such a toxin is isolated, there is nothing specific in the treatment of burn toxæmia; it is simply that of its contributing causes.

Conclusions.

It is my contention that it would be well to establish burns units in the larger hospitals of Sydney in view of the city's increasing industry. It might be necessary to concentrate burns into one or two units because of economy. At the Royal North Shore Hospital, for instance, there were only 163 burns in ten years. However, Colebrook and his associates (1948) showed that the extra cost of such a unit was offset by the lowered mortality and morbidity and the reduced period in hospital. In addition, there is less risk of forming a reservoir of infection to contaminate the surgical wards.

Colebrook's (1948) death rate in 203 children in Birmingham in the past three years was only 3%, as compared with 32% in 100 children in Toronto reported by Robertson and Boyd (1923).

Summary.

The treatment of burns is discussed with particular reference to the prevention and treatment of shock, starvation, infection and deformities. It has been shown that after the shock period is over, the key to the treatment of burns is the early epithelialization of the affected areas. The various clinical, bacteriological, hæmatological and biochemical controls are stressed.

Acknowledgement.

I wish to thank the General Medical Superintendent of the Royal North Shore Hospital, Dr. W. Freeborn, for permission to publish the material from the hospital. I also wish to thank Miss Stevens, the dietitian of the Royal North Shore Hospital, for the diet sheets.

Appendix A.

3000 Calories. Protein 200 Grammes. Semi-Solid Soft Diet.

Breakfast: 6 ounces porridge, 1 ounce dried skim milk, 10 grammes sugar, 5 ounces milk, 8 ounces orange juice, 2 eggs, 1 ounce bread, ½ ounce butter.

Morning Tea: 8 ounces milk, 1 egg, 1½ ounces dried skim milk.

Dinner: 4 ounces creamed minced white meat, 3 ounces mashed potato, ½ ounce dried skim milk, 4 ounces milk pudding, 1 ounce cream, 6 ounces milk.

Afternoon Tea: 8 ounces milk, 1 egg, 1½ ounces dried skim milk.

Tea: 2 eggs, 1 ounce bread, ½ ounce butter, 4 ounces milk pudding, ½ ounce cream, 8 ounces milk.

Supper: 8 ounces milk, 1 egg, 1½ ounces dried skim milk.

Appendix B.

3000 Calories. Protein 200 Grammes. Solid Diet.

Breakfast: 6 ounces porridge, ½ ounce dried skim milk, 10 grammes sugar, 8 ounces milk, 1 portion fruit, 2 eggs, 1 ounce bacon, 1 ounce bread, ½ ounce butter.

Morning Tea: 8 ounces milk, 1 egg, 1 ounce dried skim milk.

Dinner: 5 ounces meat, potato, 2 vegetables, 4 ounces milk pudding, stewed fruit, ½ ounce cream.

Afternoon Tea: 8 ounces milk, 1 egg, 1 ounce dried skim milk.

Tea: 4 ounces meat, 1 portion fruit, 8 ounces milk, 1 ounce bread, ½ ounce butter.

Supper: 8 ounces milk, 1 egg, 1 ounce dried skim milk.

Appendix C.

3000 Calories. Protein 200 Grammes. Liquid Diet for Tube Feeding.

2½ pints skim milk, 10 eggs, 9 ounces dried skim milk, 6 ounces lactose, 1 ounce "Aktavite", 10 ounces orange juice.

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Reports of Cases.

A CASE OF MESENTERIC VASCULAR OCCLUSION.

By CHARLES WATSON,

Nowra, New South Wales.

THIS note is submitted by one on the outer fringe of the scientific advances in therapeutics which are described almost weekly in our medical journals. Presented with a case which offered Hobson's choice between hopeless surgery and the empirical use of a potent drug about whose action one had had no first-hand experience, the writer was staggered at the result. Possibly other country practitioners, beset with a similar problem, may be encouraged to use what seems to be a form of therapy beyond the scope of

rural practice, rather than face the gloomy prognosis which the surgery of mesenteric occlusion entails, even under the best of conditions (Bintcliffe, 1947).

Clinical Record.

The patient, Miss B., aged fifty-eight years, had been under treatment by a heart specialist for eight years for the effects of mitral stenosis complicated by auricular fibrillation and for some years had been taking 45 minims of tincture of digitalis *per diem*. She had consulted her physician five days before, and he had been surprised and pleased to find that her heart-beat was again regular, after some years of auricular fibrillation.

On the evening before I first examined her, she had retired feeling particularly well. At about 11 p.m., she woke with a dull boring pain referred to the region of the umbilicus. Her bowels moved once and she vomited several times. The pain was continuous throughout the night. Early in the morning her sister prevailed upon her to call the doctor, because she thought the pain might be due to appendicitis.

When examined at 9 a.m., the patient looked pale and drawn and complained of intense constant pain, localized to the middle of the abdomen. The pulse rate was 48 per minute and regular; the blood pressure was 150 millimetres of mercury, systolic, and 96 millimetres, diastolic. On auscultation, a rough diastolic murmur was heard at the mitral area. Her temperature was 96·8° F. The abdomen was somewhat distended; no rigidity was detected; the liver was palpable and soft, and a vague mass, measuring about four inches across by two inches from above downwards, was felt in the upper part of the abdomen. The mass was very tender on palpation. Clinical examination of the respiratory and central nervous systems revealed no abnormality. No history could be elicited of previous abdominal pain, indigestion, intolerance of fats or frequency of micturition. At 10 a.m., the clotting time estimated by the capillary tubing method was found to be six minutes.

In view of the physical findings and the history of the recent return to normal rhythm of a heart which had for a considerable time been in a state of auricular fibrillation, a diagnosis of mesenteric occlusion by an embolic fragment was made.

The patient refused to be transferred to hospital, and at 12 noon 5000 units of heparin were administered by intravenous injection. At 2 p.m. a further 5000 units were administered, and this dose was repeated at 6 p.m. At this last visit, the patient said that the pain was considerably less, and on examination, the abdomen was no longer tender. The intravenous injection of 5000 units of heparin was repeated at 10 p.m. and at 2 a.m. the next day. At the time of the 2 a.m. injection, the patient felt perfectly comfortable, the abdomen was no longer tender and no mass was palpable.

The intravenous administration of 5000 units of heparin was continued throughout this day at 9 a.m., 2 p.m., 6 p.m. and 11.30 p.m., and on the next day at 8 a.m., 2 p.m., 8 p.m. and 12 midnight, and finally, on the fourth day after the onset of pain, at 8 a.m. and 6 p.m.

In addition, 300 milligrammes of dicoumarol were given on the first day and 200 milligrammes on the second day. No further dicoumarol was used, because the writer, from lack of experience with the Quick test, found difficulty in interpreting the end-point for the prolonged clotting time.

The patient was up and about on the fifth day of the illness, and her bowels moved normally. During the first two days after the onset of abdominal pain, she had been able to take only sips of water, but rapidly returned to the usual diet. She had continued to take 15 minims of tincture of digitalis three times a day except on the first day.

Comment.

A point of interest which arises from this case is that it was possible for the bowel to recover, even though heparin therapy was not commenced until thirteen hours after the embolus had presumably lodged in the wide mouth of the superior mesenteric artery. It seems likely that the occlusion was not complete, and that the action of heparin was to prevent the effects of massive intravascular thrombosis from being superadded to the initial preformed embolus (Cummine, 1949).

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LONGEVITY OF PARAPLEGICS.

By PAUL G. DANE,
Melbourne.

THE prognosis, as far as life is concerned, for patients with injuries to the spinal cord (gunshot wounds *et cetera*) resulting in complete paralegia has always been regarded as very poor, most patients dying within two or three years. It might be of interest to record two patients, both of whom have been completely paraplegic, one for thirty-two years and the other for thirty-three years.

Private B. received a gunshot wound of the spine on May 28, 1917. He has had complete paralysis of both lower limbs and of the bladder ever since with loss of sensation from the fifth thoracic dermatome. He has had a very severe infection of the bladder for many years and for the last six years has had the bladder drained. In spite of his paralysis and severe chronic cystitis, he is today fifty-two years of age in quite good health.

Private W. received a gunshot wound of the spine on July 27, 1916. He has had complete paralysis of both legs and bladder ever since and loss of sensation from the ninth thoracic dermatome. He has been in good health all these years and at present is in fair health, his main disability being mild auricular fibrillation of the heart. He is fifty-eight years of age.

These two ex-soldiers are all that remain out of ten paraplegics who came under treatment. The other eight died within three years.

The late Sir Sidney Sewell informed me that he had a patient, an ex-soldier of the first World War, with complete paralysis of both legs and bladder, who was alive and well thirty-five years after having received the gunshot wound.

The prognosis for life of paraplegics with modern treatment should be much better now than in former years.

It would be interesting to have recorded the number of paraplegics from the 1914-1918 war that are still alive in Australia.

Acknowledgement.

This information is published by courtesy of the Deputy Commissioner of Repatriation, Victorian Branch.

CARCINOMA OF THE RESIDUAL URETER FOLLOWING NEPHRECTOMY FOR PAPILLIFEROUS CARCINOMA OF THE RENAL PELVIS: A CASE REPORT.

By V. S. HOWARTH,
Gordon Craig Fellow in Urology, The University of Sydney, and the Royal Prince Alfred Hospital, Sydney.

IN September, 1947, a female patient, aged forty-seven years, underwent the operation of right nephrectomy at another hospital. Her complaint at that time was of chronic pain in her right flank and hæmaturia. The pathological examination of the kidney on removal was said to disclose a papillary carcinoma of the renal pelvis which did not invade the renal substance.

After the operation the patient complained of a constant pain in her back, and for three weeks before being examined in February, 1949, she had noticed that her urine was blood-stained.

A cystoscopic examination was carried out and a papilliferous growth was seen to protrude from the right ureteral orifice. This tumour was treated by transurethral fulguration, and a cystoscopic examination performed at a later date showed the right ureteral orifice to be thickened, but the bladder wall to be free of neoplasm.

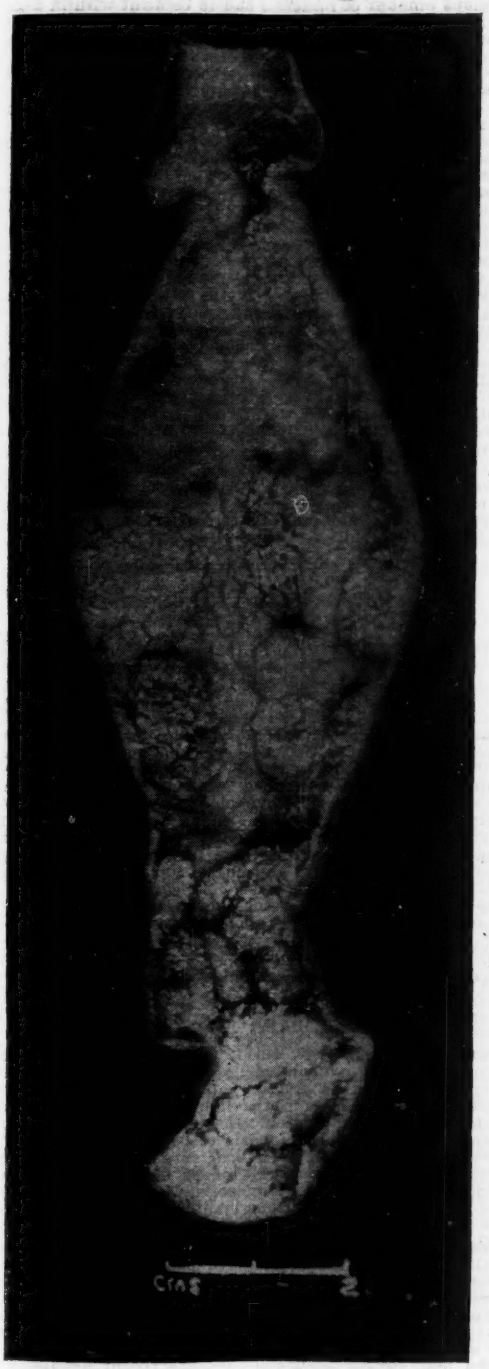

FIGURE I.
Note that the mucous membrane of the ureter has been completely replaced by neoplastic epithelium, which in places has assumed a papillomatous appearance. This latter change is most pronounced in the lower portion of the tube.

Residual juxta-vesical ureterectomy was performed after the fashion advocated by Macalpine (1947). Figure I shows the specimen removed. Examination of a microscopic section of the wall of the ureter disclosed that the transitional epithelium of the ureter had been replaced by epithelium which showed all the characteristics of *carcinoma in situ* and also papilliferous projections into its lumen.

Summary.

1. A case of carcinoma in the residual ureter following nephrectomy for papilliferous carcinoma of the renal pelvis is reported.

2. The short interval between the original operation and recurrence of the symptoms—namely, seventeen months—is noteworthy.

3. Nephro-ureterectomy in one stage is the treatment of choice for papilliferous carcinoma of the renal pelvis.

Acknowledgements.

I am indebted to Dr. H. G. Cummine, of the Department of Urology, Royal Prince Alfred Hospital, Sydney, for permission to place on record this case report, and to the Department of Medical Artistry, the University of Sydney, for the photography.

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Reviews.

THE BASIC NEUROSIS.

EDMUND BERGLER has published many papers and works during the past twenty years. He is a psychoanalytically orientated psychiatrist who has practised in Vienna and New York since 1927, being assistant director of the Psychoanalytic Freud-Clinic in Vienna and a lecturer at the Psychoanalytic Institute in New York.

His most recent publication is "The Basic Neurosis",[1] in which he attempts to prove that all types of neurosis are reducible to one common denominator—unconscious psychic masochistic attachment to the earliest image of the pre-œdipal mother.

Briefly, the outline of the theory is as follows. The child is assumed to live for some time in a fantasy of magic omnipotence, then there is a gradual understanding that fantasy and reality do not coincide owing to a series of disappointments, such as delay in receiving the breast or actual refusal. Every "refusal" produces fury because of the helplessness of the child. The object of the child's anger is mother and later father, but this aggression is inhibited and produces moral reproach and guilt. Some children take the retribution in their stride and adapt themselves to the environment, shifting their aggression to lesser objects; other children persist in the original aims with the production of punishment in the form of guilt. This latter group become psychic masochists—they are unconscious lovers of humiliation, defeat and refusal. In order that the individual can maintain the pleasure principle he must turn displeasure into pleasure—he gets libidinous satisfaction from his pain, punishment and guilt. When this aim is established in the oral stage the mother in the pre-œdipal stage is the malefactor. Later, people from whom similar pleasurable displeasure is obtained are unconsciously identified with the mother.

However, it appears that the superego disapproves of this form of pleasure, hence the ego creates new defences and makes it possible for the oral neurotic to enjoy self-pity and the pleasure of being refused.

It is apparent that the author expects opposition to and disbelief of his assumptions as he defends himself stoutly at the outset of the book, and it must be confessed that

[1] "The Basic Neurosis: Oral Regression and Psychic Masochism", by Edmund Bergler, M.D.; 1949. New York: Grune and Stratton, Incorporated. 8½" × 5", pp. 372. Price: $5.00.

his theories require considerable concentration if one is to follow them, and at times, an almost blind faith if one is to believe them. One-third of the book is devoted to the explanation of his theory, and the remaining two-thirds to twenty-seven clinical pictures of oral regression, covering a wide field of neurotic and psychopathic behaviour disorder.

On the whole, the book is an interesting treatise, although in parts difficult to read; it is even more difficult to see the connecting links in the reasoning in some of his clinical studies.

To the reader already experienced in analytical writings, the book will be stimulating, to the psychiatrist it will be controversial and to the general practitioner probably of little interest.

HISTOPATHOLOGY OF IRRADIATION.

"HISTOPATHOLOGY OF IRRADIATION FROM EXTERNAL AND INTERNAL SOURCES" is the title of one of a series of volumes prepared as a record of research work done under the Manhattan Project and the Atomic Energy Commission.[1] The name Manhattan Project was assigned by the Corps of Engineers, United States War Department, to the scientific and engineering activities which had as their objective the utilization of atomic energy for military purposes. The record of research work, when completed, is expected to consist of some 60 volumes grouped into eight divisions. The work of Division IV (known after the bombing of Nagasaki, as the Plutonium Project) includes several "health volumes", one of which is the book under review. Edited by William Bloom, with the help of ten contributing authors, it is a record of three years of intensive war research, undertaken to compare the histological changes that result from various types of radiations originating externally and internally. Animals were exposed to X rays, γ rays, fast neutrons, slow neutrons, or β rays from an external source of radio-phosphorus, or received intravenous, intramuscular, or intraperitoneal injections of radioactive material. "This work was done", writes Bloom in his preface, "by numerically insufficient scientific and technical personnel"—they began work with one histologist and a half-time technician!—"in haste and with little time to repeat experiments or to fill in gaps in the material. However, we were provided with a profusion of animals, liberal amounts of old and new radioactive materials (alpha, beta and gamma emitters) for internal administration, excellent facilities for exposing animals to beta and gamma rays and fast and slow neutrons, a co-operative spirit on the part of other investigators on the project, and facilities and personnel for determining accurately the amount of radiant energy used." The work described was carried out between April, 1943, and October, 1945. It was, essentially, a by-product of the Atomic Energy Commission. Professor Bloom and his associates were asked to undertake it, early in 1943, for three reasons: firstly, histological study of the blood-forming and other organs was necessary to explain the post-irradiation changes in the peripheral blood; secondly, it was thought at the time that the Germans might use "radio-active poisons" as a new type of chemical warfare agent; and thirdly, it was realized that if and when nuclear energy was released explosively, enormous amounts of radiant energy would be liberated. Thus histological studies became "obligatory", not only as an aid in establishing "tolerance levels" for the personnel of the atomic energy project, but also as a means of learning what might be in store for troops and civilians.

The broad outlines of their work were thus prescribed for Bloom and his associates by the much larger project of which they were a part; it was possible to perform only a few "tentative trials of additional experiments which seemed indispensable to the understanding of some of the results obtained". The main task was to examine animals killed at various intervals after treatment with one or more applications of various radiations. In general the dosages used were the "LD 50/30 days" (dose lethal to 50% of the animals used within 30 days) and fractions thereof which were decreased until morphological changes were no longer to be found. Most of the work was done on mice and rats with a relatively small number of rabbits and guinea-pigs. Many of the animals had been exposed to irradiation for purposes other than histopathology (that is, for mortality data, hæmatology, terminal pathology; this work is described in reports other than the present volume).

In chapter I, Professor Bloom describes with admirable clarity and detachment the scope of the experiments, their merits and deficiencies, and defines some of the histological terms used in the report. This was a wartime project; an enormous amount of material had to be dealt with in a given time; the *tempo* is indicated by the fact that the report was being written months before all the experimental animals in some of the series were to be killed. While some of the purposes for which the group was set up were accomplished, the authors acknowledge regretfully their failure to discover why animals die after irradiation and why certain cells and tissues are radio-sensitive and others are radio-resistant. They believe, however, that they have delineated, rather more precisely, some aspects of the problem of the histological effects of irradiation. The effects on each major organ or tissue of the body are described and very fully illustrated; of the 808 pages in this book, 416 are devoted to photomicrographs, autoradiographs, *camera lucida* drawings and diagrams, all beautifully reproduced. The chapters on bone marrow and on lymph node and intestinal lymphatic tissue are of particular importance; the regular order in which the various types of hæmatopoietic cells are destroyed is of great interest. In the section on the vascular system it is suggested that alterations in the blood vessels of irradiated animals may possibly be secondary to the course of inflammation of the surrounding connective tissue. In regard to the postulate that the most primitive cells are especially radio-sensitive, Bloom suggests that radio-resistance is not regularly a function of the primitiveness of the cell, but rather of its condition at the time of irradiation.

Altogether this book contains a vast amount of information; it is a valuable record and should prove a useful reference book. Yet it does but touch the fringe of one of the many vast fields opened up to the mind of man by the discovery of atomic energy—that great source of power which has still to be studied, harnessed and controlled.

PROGRESS IN OTO-LARYNGOLOGY.

"RECENT ADVANCES IN OTO-LARYNGOLOGY", by Dr. R. Scott Stevenson, now in its second edition, maintains the high standard set by its predecessor.[1] It makes a timely appearance, the first edition, published in 1935, now being out of print.

The advances during the intervening years have been so numerous and diverse that they have necessitated a revision of the entire subject matter. Five chapters have been eliminated and three new ones on chemotherapy, on hearing aids and on aviation oto-laryngology have been added. The result is a handy compendium which aims to "stand somewhere between a year book and a text book . . . and to assess, comment upon and come to some conclusion on a range of subjects selected as being of topical interest". Apart from stimulating the interest of medical practitioners, it should also be of use to those working for higher degrees and of particular interest to those practising oto-laryngology.

The noteworthy literature of recent years is thoroughly reviewed. But the book goes much further than that. Each chapter starts with an interesting historical survey.

Next comes the principal subject matter of the particular advance under discussion. Abstracts, annotations and references are succinctly presented.

Finally—and this will surely be an attraction to busy doctors—there is a concluding summary. From a wide experience the author gives a balanced appraisal of modern trends, not necessarily in the light of his own judgement, but in that of world opinion. In fourteen lines, for example, he can reorientate the emphasis to be placed on antibiotics, allergy, and intranasal or radical surgery in the treatment of nasal sinusitis. Similarly tonsils and adenoids, chronic middle ear suppuration, otosclerosis, Ménière's disease, deafness and cancer in its various manifestations are all brought into present-day perspective.

Conflicting views are impartially handled, a particularly gratifying feature in contentious subjects such as tonsillectomy during a poliomyelitis epidemic, otosclerosis and pregnancy, bronchoscopy in asthma, the treatment of laryngeal cancer, standardization and fallacies of audiometers, "the myth of increased bone conduction", to name but a few.

The new edition accurately reflects recent advances and merits the highest commendation.

[1] "Histopathology of Irradiation from External and Internal Sources", edited by William Bloom, M.D.; 1948. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 8" x 5", pp. 850, with many illustrations.

[1] "Recent Advances in Oto-Laryngology", by R. Scott Stevenson, M.D., Ch.B., F.R.C.S. (Edinburgh); Second Edition; 1949. London: J. and A. Churchill, Limited. 8" x 5¼", pp. 408, with 106 illustrations. Price: 24s.

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THE HEALTH OF BRITAIN IN 1947.

FROM Great Britain has come the report of the Chief Medical Officer on the state of the public health for the year ended December 31, 1947. It will be remembered that the *National Health Service Act* came into force on July 5, 1948. The state of the public health prior to the introduction of the new service would be expected to have a considerable bearing on the success or failure of that service. Any reasonable person would think that years of self-denial and restriction, voluntarily undertaken, to win a war extending over several years, would have an adverse effect on the health of the people. But the successive reports from the Ministry of Health have shown that Britain's people maintained their health both during the years of war and in the years of post-war stringency in a fashion that was a matter for continual wonder and admiration. The present report creates the same impression. It may be well at this stage to recall one fact—that the rationing of foods, though it created hardship for the housewife and others who had to stand in queues, was effective. We may recall in particular the arrangements which were made for the distribution of milk, by which children and nursing mothers received special consideration. The year 1947, the eighth year of austerity, is described by Sir Wilson Jameson, the principal medical officer, as a testing year. "Its first three months formed a winter of exceptional severity, which had to be endured by a people who in addition to rationing of food were faced with an unprecedented scarcity of fuel. These three months of snow and bitter cold were followed in April by the heaviest floods for 53 years, which did great damage, killed thousands of sheep and lambs, delayed spring sowing and threatened the prospect of a good harvest, which was so urgently needed." The four months of disastrous weather were followed by a period of economic crisis with an increasing deficiency of dollars. The crisis was so acute that restrictions more rigorous than any imposed in the war years became necessary. The meat and bacon rations were reduced and bread and potatoes had to be rationed. Conditions were grim, but the principal medical officer remarks that affairs abroad were as depressing as conditions at home. He goes on to observe that, in spite of all this, the British people "with their accustomed resilience" made a good recovery. They were in his opinion "cheered by a warm summer and in the late autumn by the enthusiasm aroused by the Royal Wedding". The resilience of the people of Britain has been shown on many occasions and factors of all kinds minister to it. A warm summer will be certain to cheer the heart of a Briton, and his make-up is such that an event like a royal wedding moves him deeply. To be present in England on a royal occasion will dispel any doubts that one may have on that score. Perhaps one may be forgiven for the suggestion that resilience is not necessarily a permanent quality in any people and that conditions calling it into evidence should be removed on every possible occasion. Resilience lost will be hard to reestablish.

But to return to our report—we note that vital statistics remained excellent and that many of the low records of mortality set up in 1946 were again lowered. The principal medical officer asserts that these statistics furnish the indisputable data upon which any real assessment of the people of England and Wales must rest. The birth rate, rising again, reached a level of 20·5 per thousand, higher than in any year since 1921. The crude civilian death rate was 12·3 per thousand; the figure was 12·0 in 1946 and 12·6 in 1945. Most of the increase in mortality was due to diseases associated with old age, malignant disease, intracranial lesions, diseases of the heart and circulatory system and all forms of respiratory disease, except influenza. Influenza during 1947 was mild in type and caused fewer deaths than in any year since 1915, except 1945. The exceptionally severe winter is regarded as having possibly something to do with the increased old-age deaths. The statistics relating to mothers and infants are striking. Although the birth rate was the highest since 1921, the infant mortality fell to 41 per thousand live births; in 1946 the figure was 43. As a sign of progress the fact is recalled that in 1921, which, like 1947, had a hot summer, the rate was 83 per thousand. The stillbirth rate of 24 per thousand total live and stillbirths showed a steeper decline than in previous years and compared favourably with the figure of 27 for 1946. The maternal mortality rate showed a further decline and was 1·17 per thousand total births as compared with 1·43 in 1946. The enlightened food policy, according to which priorities and supplements are provided for expectant and nursing mothers, infants and young children, is held to have had no small share in the continued lessening of the rates. It may be remarked in passing that further decrease in maternal deaths may be expected to occur, for reference is made to a group of cases in which emergency or transfusion services were not obtained or were not available, and to deaths from toxæmia in which ante-natal care was inadequate and "ante-natal beds" were insufficient in number. On the nutrition side we read that data were obtained in 1947 from 17 areas in England and Wales and that a slight but definite loss in weight among children was found, compared with the figures for 1945. "It would be premature to speculate as to the cause of the decline, but it should be pointed out that the rations and allowances of the school child, including school meals, are still sufficient or more than sufficient for his needs and that they have not been changed substantially since 1945."

Some figures on the incidence of certain diseases may be quoted as demonstrating the value of prevention—presumably there are people still who have to be impressed with, or reminded of, the importance of the prevention of disease rather than of its cure. For the sixth year in succession the number of deaths from diphtheria was the lowest yet recorded. The number of cases occurring in 1947 was the lowest recorded, being less than half the number for 1946. During the year 450,000 children under the age of five years were immunized, but this number was 140,000 short of the "target" figure. For 1948 the target figure is 635,000 children under five years of age. There was a fall in the number of notifications of dysentery and also in the number of deaths from that disease. Notifications of scarlet fever numbered 58,027 and the number of deaths was 42, a new low record. Reference is made to the work of Hamburger on the nasal carrier as the chief source of infection and to the fact that large numbers of patients carry *Streptococcus pyogenes* in nose and throat at the time of their discharge from hospital. In one investigation after a period of ten weeks no less than 24% of contacts carried this organism. The case fatality in measles was 0·16%, the lowest rate recorded. There were 92,662 corrected notifications of whooping-cough and 905 deaths—an apparent fatality rate of 0·98%. So far the Ministry has not advocated the combined immunization against diphtheria and whooping-cough, as it does not wish to jeopardize the success of the anti-diphtheria immunization campaign. For eight years the incidence of cerebro-spinal fever has remained high—in 1947 the notifications were twice as many as in 1937 and 1938.

Mass radiography of the chest has been extensively used. Up to December 31, 1947, 2,019,670 persons had been examined by this means in England and Wales. Of this number 94% were found to have no abnormal chest condition. Previously unsuspected tuberculosis was revealed in 7892 persons and in most cases the activity was proved at once. A total of 257 cases of intrathoracic malignant disease were discovered. The position in regard to "B.C.G." vaccination is interesting. Arrangements have been made for "B.C.G." vaccine to be made available in each region to tuberculosis specialists wishing to administer it on their own responsibility. "The intention is not to encourage general inoculation of the public at large, but in the first instance, to concentrate on those groups considered to live at more than average risk of tuberculous infection, because it is realized that no certain claim can yet be made for this type of inoculation in the conditions prevailing in this country." The criticism seems justified that excessive caution is being shown and that at least, in view of the results obtained in Scandinavian countries, an investigation to determine the value of "B.C.G." in British conditions should have been made long ago.

There are many other aspects of the report which would well repay examination and discussion. The present object has been to show that the health of England and Wales need not be in any doubt. An amazing result is being achieved and the greatest credit is due to Sir Wilson Jameson and those with him who have planned the activities of the Ministry of Health. The Ministry of Health could not have done what it has done if the way had not been prepared by the expert care that has been displayed in the rationing of food and in the preparation of the community for the load it has had to carry. Those who have charge of the nation's health have to deal with conditions as they find them. In the Old Country the "resilience" already mentioned has without doubt been the nation's salvation. This resilience shows itself readily in the field of medicine—public health and preventive medicine. It will be of the greatest interest to dissect next year's report and to look for possible effects of the newly introduced national health service.

Current Comment.

ACUTE MASTITIS.

ACUTE MASTITIS is a common enough condition, but not much has been written about it in recent years, despite the advances in its treatment that have come with the sulphonamides and antibiotics. A practical consideration of the subject has been made recently by Anthony Walsh[1] in a paper based on the study of 153 cases of acute mastitis treated between December, 1945, and January, 1947. Walsh's main concern is with treatment, but his principal findings in relation to ætiology should be mentioned. The point which impressed him most was the significance of milk stagnation, and from this he suggests that it must be of paramount importance in treatment to ensure that the breast is properly emptied of milk and kept empty; this principle was supported by his results. The accepted predominance of *Staphylococcus aureus* as the principal bacterial agent concerned was confirmed; a pure culture of hæmolytic streptococci was grown in one case, and in a small number of cases the streptococcus appeared to be a secondary invader. The onset in the great majority of cases was after the first week of the puerperium, and Walsh points out that over half the cases were found at a time when the patient would not normally be in hospital; hence the responsibility for early diagnosis and treatment often rests with the general practitioner. The main subdivision of treatment is related to the presence or absence of abscess formation. In the absence of abscess formation, in all cases the administration of penicillin at the earliest sign of inflammation is most important (a delay of as little as twelve hours makes a difference); the dosage used in the series was 20,000 units given intramuscularly every four hours, and this appears to have achieved useful results, but Walsh mentions that since completion of the series he has adopted a dosage of 100,000 units every four hours, "for some strains of *Staph. aureus* are relatively less sensitive to penicillin". If the diagnosis is in doubt, Walsh urges that treatment be given. Lactation was suppressed in the early cases of the series, but this practice was soon abandoned; milk flow was then encouraged, feeding from the uninfected breast was continued in unilateral disease, and the infected breast was completely emptied after each feed by manual expression. The expressed milk, after being boiled for five minutes, was given to the infant, and no ill-effects followed this practice or the feeding from the uninvolved breast; Walsh hesitates to support the practice advocated by certain other authors of allowing the infant to feed directly from the infected breast—a cautious attitude which seems justified despite claims made that the practice is harmless. The other main point in treatment is the provision of support for the breast—significant not only in the patient's comfort but also, it would seem, in the response to treatment. In the cases in which abscess formation had occurred or commenced, the principle that penicillin is not a substitute for surgery was found to hold. The temp-

[1] *The Lancet*, October 8, 1949.

tation to administer sulphonamides or antibiotics and to wait for the abscess to become "localized" should be resisted. It is not always easy, of course, to recognize the early signs of pus formation and many patients do not seek advice at this stage, but Walsh urges that any area of tense induration in an inflamed breast should be regarded as containing pus and drained accordingly; apart from its ultimate therapeutic value this practice affords the patient great relief from pain. In cases of mastitis which has lingered on for weeks under insufficiently vigorous treatment and in which there is a zone of induration that is only moderately tender, further observation for two or three days in conjunction with adequate penicillin therapy (50,000 to 100,000 units every four hours) is justified. Walsh used the classical radial incision at first and then tried a curved incision in the submammary fold, but found both unsatisfactory. He then adopted, with gratifying results, an incision about one centimetre long placed exactly along the areolar margin. To avoid duct damage the incision is made through skin and superficial fascia only, and through it a long pair of narrow-bladed artery forceps is thrust into the abscess cavity. By palpation on to the instrument with the left hand laid flat on the breast, all indurated areas and loculi, it is stated, can be entered and drained. As much as possible of the pus is expressed, a small rubber tissue drain is inserted for twenty-four hours, a dry dressing is applied, and the breast is so bandaged as to keep it well supported. Penicillin, 100,000 units every four hours, is given systemically, the first dose being administered with the pre-operative atropine injection. Emphasis is laid on manual expression of milk from the infected breast, as in the other cases. The effect of this incision in Walsh's series, from the point of view of recovery, healing and cosmetic result, was very satisfactory, and he recommends it for any abscess near to the areola. In the few cases in which the abscess is near the periphery or in the axillary tail, he considers that the same general technique can be followed, a small tenotomy incision being made over the point of maximum swelling. Walsh had three aims in this investigation of the treatment of acute mastitis. The first was rapid control and cure: it appears that these can be achieved by the systemic administration of penicillin if treatment is started at the first sign of inflammation, and he firmly believes that if all cases of acute mastitis are so dealt with, no breast abscess need occur. The second aim was minimal interference with breast-feeding: in the series not only was weaning unnecessary, but the disease was found to be easier to control when the flow of milk was maintained. The third aim was minimal scarring when drainage was required: the incision described leaves an almost invisible scar and apparently provides adequate drainage with minimal damage to breast tissue. The achievement of these aims suffices to commend the paper.

UNDECYLENIC ACID AND PSORIASIS.

A PRELIMINARY report by H. H. Perlman on the effects of treatment of a small series of patients with chronic psoriasis by means of undecylenic acid was mentioned in these columns on July 30, 1949. Perlman made no claims at all in his paper, but his results certainly commanded attention and a more recent report must be viewed with interest. In this Perlman and I. J. Milberg[1] describe the effects of undecylenic acid on 41 patients with chronic psoriasis at the New York Skin and Cancer Clinic. The majority of the subjects chosen were psoriatic patients whose lesions had proved refractory to other recognized forms of therapy, or who had not had spontaneous remissions for a substantial period of time preceding the new approach. All external medications were excluded. Dietary and other habits prior to the period of treatment remained essentially unchanged. The undecylenic acid was given by mouth. Serious or lasting toxic effects were not observed; mild gastro-intestinal disturbances occurred, but were sufficient in only one case to stop further treatment. Of 40 patients with cutaneous lesions, 12 showed unequivocal improvement during treatment, 15 were somewhat improved, 10 showed no change and three became worse. High standards were set for the grading as "improved" and "somewhat improved". Particularly in the improved group, some pattern in the process of involution could often be discerned: relief of itching, flaky desquamation, flattening and paling of the inner part of the plaques and the appearance of clinically normal, usually somewhat hyperpigmented, skin. In two cases in the "improved" group complete freedom from lesions was observed after two and a half months; the administration was continued in both cases for one month after the clearing of the lesions without any recurrence and then stopped. In two other cases, in which treatment was discontinued a few weeks after improvement had started, the psoriasis once more became aggravated and new papules appeared; with resumption of treatment, improvement was noted again. Seven of eight patients with psoriatic arthropathy noted relief from pain. Perlman and Milberg are most emphatic in refusing to make therapeutic claims for the method, having in mind the unpredictable course of psoriasis and its tendency to "spontaneous" remissions, as well as the smallness of their series. However, their results appear to justify amply the very mild statement that it would "appear somewhat unwise to assume that these results were all merely coincidental". It is to be hoped that other dermatologists with adequate facilities for controlled investigation will join in this study.

[1] *The Journal of the American Medical Association*, July 9, 1949.

AUREOMYCIN AND CHLOROMYCETIN.

ONE of the most curious results of the outstanding success of penicillin has been the emergence of other antibiotics, which have so soon proved their right to extended therapeutic trials that the medical world can as yet hardly distinguish established methods from those of promise. Once again we think of the aphorism that a new drug takes ten years to test thoroughly, and wonder when we shall catch up with these by-products of organisms which are displaying so unsuspected a benignity. Aureomycin, derived from a member of the genus Streptomyces, isolated from the soil, resembles in its origin chloromycetin, though the physical and chemical properties of the two substances are different. Both have the great advantage that they may be effectively administered by mouth, though aureomycin often causes some nausea and vomiting. Fortunately, neither animal experiment nor therapeutic trial indicates that either drug has any important toxic effects. Details of the absorption characteristics of both are readily available in the literature, which is now becoming voluminous. What interests medical practitioners is their clinical effectiveness. This is not easy to assess till considerable tral has been made, but one impressive feature of both drugs is a wide range of usefulness. The work of the last few years has shown that at last we appear to have drugs which are in some degree specific for rickettsial and viral infections. Penicillin, of course, has been limited in this regard, and even its power to destroy or neutralize the *Treponema pallidum* should be viewed with caution, as recently pointed out by Molesworth and Cooper Booth in these pages. M. Finland, H. S. Collins, T. M. Gocke and E. B. Wells have published a fairly comprehensive review of the present position of aureomycin, and in this they lay emphasis on the wide range of infective agents against which the drug appears to be potent.[1] Experimental findings do not run parallel with clinical results, but in any case results of in-vitro experiments cannot be applied to human conditions with any certainty. Good results have been obtained from treating patients with *lymphogranuloma venereum*, and more important, with rickettsial diseases, in particular typhus fever. Chloromycetin has been used also with great success in all the varieties of typhus, and one sig-

[1] *Annals of Internal Medicine*, July, 1949.

nificant property of these drugs is that they have given good results even when the infection was well established. Instances of this are given in a comparison of the results obtained with aureomycin and chloromycetin published by T. E. Woodward.[1] Both these drugs give better results, with less risk of side effects than para-aminobenzoic acid. The effects on "Q" fever are less striking, and so far cannot be considered as established. One of the most interesting results recorded is in primary atypical pneumonia. Though this is believed to be due to a virus, its nature is less clearly understood than the agents of infections which cause similar forms of pneumonitis, such as the rickettsial diseases, tularæmia, influenza and the ornithoses. Therefore careful differentiation is necessary, and as yet the number of cases in which treatment has been carried out is small. However, Finland and his co-workers state that aureomycin is the first therapeutic agent whose influence on atypical pneumonia has been regularly beneficial. It seems that Woodward is justified in stating that these two antibiotics are comparable with penicillin in their therapeutic powers.

One more disease may be mentioned, which has become a touchstone for the antibiotics as they have come over the horizon. This is brucellosis, which has been found to respond favourably to a combination of streptomycin and sulphadiazine. Relapses have occurred, with a resurgence of the brucella in the blood-stream. Such relapses have been treated successfully with the new drugs. However, it must be admitted that these, too, have not been competent to prevent relapse in all instances in which they were used from the first. Perhaps this is a question of length of treatment and dosage. The results of treatment of typhoid fever with aureomycin have been reviewed previously in these columns. While it appears that this drug has been successful in at least half the patients treated, its action has been irregular, and it has not always been able to eradicate the organism. Chloromycetin now appears to be the drug of choice for enteric infections. Whether the future will see an array of antibiotics facing the physician, who will have to select the appropriate drug in each instance, or whether a few master keys to the intricate locks of infectious disease will be found, cannot be foretold. Already, however, we seem to be passing from the period of empiricism to one of more guarded appraisal.

Though the toxic properties of all the antibiotics so far introduced into clinical medicine seem to be slight, we cannot assume this to be true in the future, and in any case, there can be no question of the necessity of understanding the physical and chemical properties of the drugs which research has put into hands that have been only too often impotent in the past.

MUMPS ORCHITIS.

ORCHITIS is a very undesirable feature of mumps amongst men—it is hardly ever seen in children—and by no means rare. Its incidence varies considerably from one epidemic to another, but, according to A. L. Hoyne, J. H. Diamond and J. R. Christian,[2] an average of approximately one out of every five men affected with mumps experiences testicular involvement. Atrophy is said to occur in over half the cases, the accompanying pain can be very severe, and the period of illness with loss of working days may be considerably increased. It is not surprising then that many measures, both surgical and medical, have been tried in its treatment and attempted prophylaxis. Hoyne, Diamond and Christian have reviewed the literature on the subject and they discuss the findings of other authors. Strict bed rest appears to have no prophylactic value. The results of prophylactic administration of convalescent serum are conflicting. Little or no benefit appears to have been obtained from pooled normal plasma. Results obtained with convalescent serum γ globulin indicate a high prophylactic efficiency, but pooled plasma γ globulin seems to be of little worth; available information, however, suggests that the value of convalescent serum γ globulin may be limited, and it is not always readily available. In the treatment of established orchitis, apart from obvious symptomatic measures, incision of the *tunica vaginalis* has been repeatedly advocated, particularly for the relief of pain, but also for shortening of the period of disability and prevention of atrophy. A much simpler measure is advocated by Hoyne *et alii*—the injection of diethylstilbœstrol. Its use was suggested by the facts that orchitis rarely occurs before puberty and that diethylstilbœstrol is known to decrease the activity of the testes and to cause them to revert to the pre-pubertal type of gland. Reference is made to another series of 77 patients treated by Savran; only three of these developed orchitis when diethylstilbœstrol was given in adequate dosage, in contrast with 28 cases of orchitis amongst 168 controls. Toxic effects were lacking. Hoyne and his colleagues claim similar results though their series was smaller. For prophylaxis two milligrammes per day appeared to be an effective dose. Therapeutically five milligrammes were given daily and the results among 19 patients were striking; relief of symptoms was dramatic in some cases, and in every instance the response was good with an uneventful recovery, the average duration of treatment being 4·73 days and the average total hospital stay 5·89 days. Hoyne and his colleagues express enthusiasm for diethylstilbœstrol in both prophylaxis and treatment, and they may be right; it is certainly a simple and relatively cheap measure and the results they report are attractive. Just the same it may be better to wait for others to reproduce their results before judgement is passed.

[1] *Ibidem.*
[2] *The Journal of the American Medical Association*, June 25, 1949.

THE DANGERS OF INTRATHECAL MEDICATION.

EVERY little while a controversy arises somewhere regarding the injection of drugs, sera, anæsthetic agents *et cetera* into the intrathecal space. The recorded ill effects are matched against the value of the particular route of administration, each is magnified or diminished according to the individual viewpoint, and the argument gradually subsides. It is really as well that this controversy should not be allowed to die completely, for the two opposing viewpoints are both important; it is most unlikely that general agreement will be reached at present that both intrathecal medication and spinal anæsthesia can be dispensed with; on the other hand it is most undesirable that an attitude of complacency or a false sense of security should ever develop. For this reason, though the subject is not new, we would draw attention to an appreciation of the dangers of intrathecal medication by George Wilson, Charles Rupp and William W. Wilson,[1] from the department of neurology and laboratory of neuropathology of the Philadelphia General Hospital. They review the literature and quote from their own experience to show that in many cases neurological complications follow the intrathecal injection of drugs, anæsthetic agents and antibiotics, the residual effects being often permanent, disabling and not amenable to any known therapy. Serious effects have resulted from the intrathecal injection of tetanus antiserum, meningococcal antiserum, convalescent poliomyelitis serum, alcohol, spinal anæsthetics, sulphonamides, penicillin and streptomycin. This fact must never be allowed to sink out of sight, and prevention of these unhappy sequelæ must be sought. As Wilson and his colleagues put it, consideration must be given to whether intrathecal administration is necessary and, if it is, whether the benefits derived therefrom outweigh the potential hazards. In regard to spinal anæsthesia, they rightly stress that "the gravity of spinal arachnoiditis and subsequent paralysis must enter into the meditations of surgeons and anæsthetists when determining procedure". An adequate neurological history and examination should be completed for every patient considered for spinal anæsthesia, and another anæsthetic agent should be chosen if any abnormalities, congenital or otherwise, are found, or if a history of previous neurological disorder

[1] *The Journal of the American Medical Association*, July 30, 1949.

is elicited. In the matter of administration of therapeutic agents, Wilson *et alii* write more strongly, but surely with justification. They state that when adequate spinal fluid concentrations of an agent can be obtained after systemic administration, as with the sulphonamide compounds and penicillin, intrathecal administration is "truly meddlesome mischief" and is strictly contraindicated. They concede that more extensive studies are indicated to determine definitely whether or not adequate therapeutic concentrations of streptomycin are obtained in the spinal fluid from systemic administration, and suggest a controlled study in which alternate patients should not receive intrathecal medication. It is pointed out that the hæmato-encephalic barrier is diminished in disease of the nervous system, and that adequate diffusion may occur under pathological conditions even though permeability is inadequate in normal experimental subjects. Another point is that the spinal fluid reflects only what is going on in the adjacent neural tissues. The infection is in the meninges and neural parenchyma, which have an adequate blood supply. Diffusion of therapeutic substances through the subarachnoid space is slow, and many regions of the nervous system are relatively inaccessible. Since the usual direction of flow of cerebro-spinal fluid is from the ventricles to the subarachnoid space, it is doubtful, Wilson and his colleagues consider, that an appreciable quantity of any medicament introduced into the lumbar sac diffuses to any extent through the subarachnoid pathways. Probably, they suggest, any benefit from therapeutic substances injected intrathecally occurs only after absorption into the blood-stream has occurred. Their summing-up is that "the use of this portal is illogical for most therapeutic agents and may be unnecessary for all". While a patient should not be deprived of a definite therapeutic benefit without good reason, the risk of grave complications must always be weighed against possible advantages.

BIRTH WEIGHT AND PHYSICAL DEVELOPMENT.

THE family doctor who is not equipped with information about the healthy child is liable to lose face, no matter how capably he may manage when children are ill. An accurate or inaccurate prophecy of, for example, the appearance of a tooth can make or mar him in the parental eye. One subject in which parents are interested, but on which there has been little reliable information, is the relation of birth weight to physical development in childhood. Most of the studies reported relate to premature infants, the usual finding being that they tend to remain below normal size; but even these reports are not all conclusive. An interesting and quite extensive study carried on for a period of over ten years was started from the observation in the out-patient department of the Hospital for Sick Children, Great Ormond Street, London, that a notable proportion of children who were below the average weight had been small babies at birth. The results have been reported by R. S. Illingworth, C. C. Harvey and Shan-Tah Gin.[1] A study was made of the weights and heights at various ages of 517 children who at birth weighed five and a half pounds or less, of 712 children who at birth weighed from seven pounds two ounces to seven pounds six ounces, of 940 children who at birth weighed from eight and a half pounds to nine and a half pounds, and of 257 children who at birth weighed nine pounds nine ounces or more. It was found that at all ages the weight at birth bore a well-marked and constant relationship to subsequent physical development. Among girls, for instance, there was a difference of 11·2 pounds at the age of seven years between the mean weight of children who had weighed nine pounds nine ounces or more at birth and that of children who had weighed five and a half pounds or less; there was a corresponding difference of 11 pounds at the age of nine years and 12·8 pounds at the age of eleven years. The average girl aged ten years who had weighed five and a half pounds or less at birth weighed the same as the girl aged seven and a half years who had weighed nine pounds nine ounces or more at birth. At all ages there was a constant gradation in the mean weight of children of either sex according to birth weight.

A strikingly high proportion of children of the smallest birth weight were below the "normal" weight in later childhood (the applicability to all children of the so-called normal standards is questioned by Illingworth and his colleagues and may be clarified in future studies). At the age of nine to nine and a half years, 41·4% of the children were 10% or more below the "normal" weight, compared with 7·8% of children of the largest birth weight; the corresponding figures for the age of eleven to eleven and a half years were 47·4% and 7·1%. At the age of nine to nine and a half years, 15·7% of 140 children of the smallest birth weight were 20·1% or more below the "normal" weight, compared with about 1% of 554 children of the remaining birth-weight groups.

There were similar but less striking changes in the mean height of these children at all ages. The main factors responsible for these changes in height and weight are not yet clear. Illingworth and his colleagues do not seem impressed with the theory that the changes are related to malnutrition of the babies of small birth weight and are investigating the matter further. They suggest that if malnutrition is not the significant factor, new developmental charts should be constructed to make it possible to assess the normality of development of the child in relation to his birth weight. Meantime we wonder if the family doctor feels much wiser. This study, like so many others of its type, deals with the "average" child of each group. It is clear that individuals of each group do not conform to the average pattern. In every age group in this study there is an overlap of the birth-weight groups, so that in every case individuals of the lowest birth-weight group have come to weigh more than individuals of the highest birth-weight group. The general conclusions are convincing and really do supply useful information, but the wise family doctor will, no doubt, apply them with discretion to the individual child, knowing the reluctance of most parents to agree that their child is "average".

[1] *The Lancet*, October 1, 1949.

THE CONTROL OF CORTISONE (COMPOUND E).

MUCH interest has been aroused in the adrenal cortical steroid cortisone or compound E, with which promising results have been reported by Hench and others in the treatment of rheumatoid arthritis (see THE MEDICAL JOURNAL OF AUSTRALIA, October 1, 1949, page 509). Cortisone, which was first isolated by E. C. Kendall, was originally obtained from the cortex of the adrenal gland, but is now being prepared from a bile acid. The amount of the substance at present available is small and it will be generally agreed that it should be distributed in such a way as will aid the elucidation of important aspects of its use not yet understood. It has now been announced[1] that a Committee on the Investigation of Cortisone has been appointed, with the sanction of the council of the National Academy of Sciences, at the request of the Research Corporation. The Research Corporation, a non-profit organization in the United States which administers patents in the interest of public welfare and for the furtherance of scientific research, has aided in the development of the processes for making cortisone, and will continue to further this development and to stimulate research in the mechanisms of action of the substance. The Research Corporation has given an assurance that the recommendations of the committee will be accepted as final with respect to distribution of the available supply of cortisone during 1949. Applications for a supply of cortisone must be submitted on a form that can be obtained from the chairman of the committee, Dr. Chester S. Keefer, 2101 Constitution Avenue, Washington 25, D.C. It is made clear that consideration will be given only to requests from institutions where adequate facilities for investigation and clinical control are available.

[1] *The Journal of the American Medical Association*, August 6, 1949.

Abstracts from Medical Literature.

DERMATOLOGY.

Epithelioma Arising in Lupus Erythematosus.

G. E. W. WOLSTONEHOLME (*The British Journal of Dermatology and Syphilis*, April, 1949) states that when an epithelioma arises in skin affected already by *lupus erythematosus*, an attempt should be made to answer three questions put by Keutzer in 1925: Is the combination accidental? Is the epitheliomatous change initiated by treatment given for *lupus erythematosus*? Or do the features of the case support the hypothesis that *lupus erythematosus* itself predisposes to cancer? The author reports a case in which epithelioma arose on the site of long-standing *lupus erythematosus*. According to Durand, epithelioma has not been recorded in combination with acute or subacute *lupus erythematosus*; it is found usually on the oldest lesion of *lupus erythematosus*, and tends to appear where there is dense scar tissue. This is probably the reason for the slow growth and late adenopathy of these tumours, whatever their cell type. Histology of the growths in 54 specimen biopsies in the literature has been reported by Beeson and Elbert to be prickle cell in type in all but two, one being basal celled and the other mixed basal and prickle celled. The treatment for *lupus erythematosus* has often been blamed, especially radiotherapy. Apart from the fact that cases were reported prior to 1903-1904, when X rays were given for *lupus erythematosus*, Durand found that such treatment had been given in not more than 35% of his cases; in 10 cases treatment of any sort had been negligible or nil. In the case reported treatment had mainly been with gold injections, "Stavarsol" and quinine. Local treatment was with calamine lotion and could have hardly been more bland. The author considers that the appearance of epithelioma in combination with *lupus erythematosus* may be accidental or due to predisposition of the tissues to cancer by *lupus erythematosus*. It is unlikely that it was caused by treatment given previously to the *lupus erythematosus*.

Herpes-like Eruption Treated with Smallpox Vaccine.

L. E. SAVITT AND S. AYRES (*Archives of Dermatology and Syphilology*, June, 1949) report a case which corresponded in many ways to one of *impetigo herpetiformis*. They state that the lesions were located in the scalp and neck, the upper portion of the chest, the back, the lips and the buccal mucosa, and consisted essentially of grouped vesico-pustules that became crusted and spread in a circular manner. Clearing of individual patches occurred spontaneously. Incomplete generalized clearing occurred at intervals of about six weeks. Recurrences of the eruption were limited in most instances to areas above the middle part of the chest. Amongst the various treatments tried, which were ineffective, were: applications of dilute dalibour water, or 5% boric acid ointment, injections of liver extract, autogenous staphylococcus vaccine, sulphadiazine given by mouth, intravenous injections of calcium thiosulphate, sulphamerazine given by mouth, pyridoxine hydrochloride given by mouth, pyribenzamine, vitamin *A* and penicillin injections. Intradermal injection of the contents of one capillary tube of a commercial smallpox vaccine was started on January 16, 1947, and repeated at intervals of two to four days. The first definite improvement was noted on February 10, three days after the seventh injection. There was no reaction to the first injection, but a vaccinial type of reaction followed the second injection. Subsequent reactions decreased in severity; consequently the contents of two capillary tubes of vaccine were injected each time. On March 8, after the fourteenth injection and fifty-one days after the first injection, the skin was entirely clear. Injections were stopped. Nineteen days later a few vesicles appeared about the face and upper gums. There was a twenty-four day interval between the fourteenth and fifteenth injections, thereafter only a one-week interval, and subsequently a two-week and then a three-week interval. The last lesion, a single vesicle, occurred on May 9, 1947. Up to the time of writing the authors had given forty-two injections, the last one on December 26, 1947, when the skin was entirely clear. The technique used is as follows: approximately 0·1 millilitre of an isotonic solution of sodium chloride is drawn into a tuberculin syringe with a number 24 or 25 hypodermic needle attached, and is then mixed in the syringe with the contents of one capillary tube of smallpox vaccine; the intradermal injections may be made anywhere, but a site near the deltoid muscle is preferable. The unknown mechanism of response to therapy in the reported case probably parallels that occurring in *herpes simplex* and apthous stomatitis and the protection afforded in smallpox.

Balanitis Xerotica Obliterans.

D. G. WELTON AND P. NOWLEN (*Archives of Dermatology and Syphilology*, June, 1949) state that *balanitis xerotica obliterans* is a chronic progressive, atrophic, sclerosing process of the *glans penis* and prepuce leading to urethral stenosis. The disease may occur at any age between twenty-two and sixty-seven years, it is not necessarily associated with circumcision or any other operative procedure, and it may be accompanied by lesions on the body characteristic of *lichen sclerosus et atrophicus*; the histological features of *lichen sclerosus et atrophicus* are identical with those of *balanitis xerotica obliterans*. Meatal stenosis occurs in many cases. Other conditions frequently noted in these cases are a constricting band of the prepuce, sclerosis of the frenum and recurring erosions and fissures of the glans. In the authors' case careful endoscopic examination of the anterior portion of the urethra did not show any evidence of a sclerosing process proximal to the meatus. Examination of the external genitalia revealed on the glans two white, dry, glazed areas of atrophy around the lateral and anterior portions of the external meatus, the atrophic plaques covering approximately one-third of the glans. The atrophic lesions were "crinkled" with an irregular border. On palpation they felt like thin parchment paper. The ventral half of the meatus was involved. On each side of the meatus were several irregular, reddish-purple, telangiectatic areas. Around the residuum of the frenum several erosions were noted; and the entire inner surface of the prepuce was thickened, inelastic and of a dead-white colour. No associated lesions in the genito-urinary tract were found. *Balanitis xerotica obliterans* is a disease of interest to urologists as well as dermatologists. Meatal stenosis, with impairment of the urinary stream, or pain on intercourse, is more likely to make the patient seek treatment than is a simple lesion on the *glans penis*.

Solid Carbon Dioxide Therapy for Cystic Acne.

C. S. WRIGHT AND E. R. CROSS (*Archives of Dermatology and Syphilology*, June, 1949) state that when using cryotherapy or carbon dioxide slush, as recommended by Eller and Wolff in 1941, for treatment of recalcitrant cases of acne, they found that the cystic lesions would almost invariably disappear after a few treatments; so they conceived the idea of using simply a pencil or solid block of carbon dioxide or dry ice for the treatment of cystic acne. The results in the treatment of more than 2000 patients with cystic acne have been so good that the authors have given up the surgical treatment of deep-seated acne lesions, which they feel increases scar formation. The application of the solid carbon dioxide may be made directly to the acne pustule, moderate pressure being used, for from three to five seconds; or if the acne lesions are numerous, a large block may be applied over a group of lesions at one time. Since the lesions are elevated, the intervening areas of skin are untouched unless the pressure is too firm. Within a few hours there may be surface vesiculation over the pustule, a development which should always be explained to the patient. This is followed by drying and a general shrinking of the treated pustules, which will usually result in their involution in from one to two treatments, with little or no scarring. Deep cystic lesions may require a number of treatments, depending on their depth.

Kaposi's Varicelliform Dermatosis.

T. ANDERSON (*The British Journal of Dermatology and Syphilis*, May, 1949) states that a clinical syndrome is recognizable of which the following are the main diagnostic features: (i) The condition attacks persons who are already suffering from or have suffered recently from some form of eczema or dermatitis. (ii) The condition has usually a sudden onset and is associated with high fever and severe prostration. (iii) The disease under suitable conditions is communicable to other persons with eczematous skin conditions and outbreaks have occurred in dermatological wards. (iv) The individual lesions consist of a vesicle which rapidly matures to a pustule; that papules occur in the earliest stage is probable, but in the great majority of cases the evolution to vesicle takes place within the first twenty-four hours, and pustulation follows with great rapidity. (v) From illustrations which accompany many of the reports it is clear that "varicelliform" is not always accurate as a pictorial description: in the first place the lesions are similar in size, although the coalescence of neighbouring vesicles may eventually result in great variation; secondly, the lesions are usually circular at the base

and may be dome-shaped or show umbilication; thirdly, the eruption all comes out at the one time so that the lesions on different parts of the body are all at the same stage of development. The author describes five undoubted cases. In reviewing the possible ætiological agents, he states that such cases might be grouped in four main categories: (i) those in which infection is exogenous and arises from contact with some other person suffering from *variola major, variola minor,* vaccinia or even perhaps varicella; (ii) those in which infection is endogenous and arises from prior performance of vaccination on the patient himself; (iii) those in which infection arises from an endogenous or exogenous source of herpes virus; (iv) those in which infection cannot be traced to any of the above sources and is presumably due to some other virus. For the condition to arise at all some preexisting dermatosis is essential, and it is desirable to stress that virus pyodermia may be in fact an infectious disease, so that strictest precautions must be taken to prevent contact of eczematous patients with any of the "pock" viruses and in particular with vaccinia or herpes. It is therefore important that as further cases are encountered they should be most closely investigated from the point of view of a virus ætiology. The material necessary for such studies should include fluid from vesicles or pustules (submitted fresh for virus isolation), the scabs from lesions (for complement fixation tests), and two specimens of blood serum, one obtained during the early stage of the illness and the other in convalescence, ten to fourteen days later, to demonstrate the development of virus neutralizing antibodies. As regards the mode of infection, especially in vaccinial cases, it seems likely that the virus is "excreted" into the traumatized areas from a general blood infection. Probably there is general dissemination of the virus and localization of the lesions is dictated by the previous skin injury.

UROLOGY.

Uretero-Sigmoid Anastomosis.

R. M. NESBIT (*The Journal of Urology,* April, 1949) presents a preliminary report on a technique designed to eliminate the principal causes of stricture at the site of uretero-colonic anastomosis. He points out that direct mucosal anastomosis between ureter and bowel is by no means a new idea. However, in the past, surgeons who experimented with the method all found that healing at the suture line was prevented by infection with consequent leakage, and the operation was abandoned. Agents now available for combating infection have encouraged revaluation of this discarded procedure. The evidence secured by now from animal experiments, as well as from operations on man, indicates that direct end-to-side anastomosis of the slit and elliptically ended ureter to the incised sigmoid colon is feasible, and not followed by stricture formation. Long-term observations of transplantations done in this way will be necessary in order to answer the question as to whether valve-like oblique insertion of the ureter is a physiological necessity in protecting the upper part of the urinary tract from infection. The author has performed two such operations on the human being, and three more operations have been performed by others in his clinic. There was no mortality, and hardly any morbidity, and there was in every case a good output of urine from the bowel soon after operation. The anastomosis is carried out bilaterally at one sitting if necessary. After one week's bowel preparation a mid-line incision is made and the peritoneum opened. The right ureter is transplanted first. It is cut across near the bladder, and the end to be transplanted is slit longitudinally for 1·5 centimetres, the end being fashioned elliptically. A longtidinal incision is now made in a suitable *tænia* coli of the sigmoid, its length being 1·5 centimetres also, and it goes right into the bowel lumen. Direct anastomosis of ureter to colon is made by a single continuous line of suture which penetrates all coats of both canals. Fine zero chromicized catgut on an atraumatic needle is used. The site of anastomosis in the colonic wall is attached to the parietal peritoneum in order to immobilize the colon in a position that will preclude angulation of the ureter. Also the extra protective covering of peritoneum aids healing. The left ureter is similarly dissected from its retroperitoneal bed and anastomosed to the sigmoid at a higher level. The abdominal wall is closed without drainage and a large rectal tube is left indwelling.

Micturition: A Fluoroscopic Study.

S. R. MUELLNER AND F. G. FLEISCHNER (*The Journal of Urology,* February, 1949) have made a special study of bladder behaviour in health and in disease, by direct observations of the action of the organ, by means of fluoroscopic study. The organ is filled with an opaque solution and the standing patient directed to urinate while the bladder is screened. The results of such observations have been very important, as they are startlingly different from those obtained by cystometric study. Cystometry led to the conclusion that micturition was initiated by direct action of the will or the vesical detrusor. As this muscle is of the plain or involuntary type, it has been difficult to understand this statement. According to cystometry, when the will has induced the detrusor to contract, the internal sphincter is opened reciprocally, followed by a reflex opening of the external sphincter. Furthermore, in voluntary stopping of the stream, cystometry indicates that it is the external (voluntary) sphincter which is contracted first. Fluoroscopy shows that the immediate effect of the will to urinate is not to contract the detrusor, but to depress the bladder base and to pull the internal sphincter downwards; it is only after a distinct lag that the detrusor is seen to spring into action. To shut off the stream, fluoroscopy shows that the effect of the will is to elevate the bladder base and therefore to stop micturition at the level of the internal sphincter. The mechanism which pulls the bladder base down, or lifts it up, is obviously located beneath the bladder that is on the pelvic floor. Study of muscle action by anatomists makes it quite clear that it is the pubo-coccygeus, aided perhaps to a small degree by the deep transverse perineal muscle, which plays this important role. In the female the pubo-coccygeus is in direct relation to the floor of the bladder; in the male the prostate gland is interposed between the bladder and this muscle, but the prostate moves up and down with the bladder base. It is for this reason that the pubo-coccygeus is sometimes called the *levator prostatæ.* At the beginning of urination, the descent of the bladder base is caused by voluntary relaxation of the pubo-coccygeus, aided by a voluntary muscle increase in intra-abdominal pressure. In the stopping of micturition the pubo-coccygeus is actively contracted, this lifting the bladder base. The authors state that when it had been demonstrated that a voluntary mechanism initiated and stopped micturition, it then became desirable to know how the act would be altered if the voluntary mechanism were eliminated. In the case of advanced carcinoma of the prostate the growth fixed the gland (and therefore also the bladder base) in the pelvis. This bladder base can be neither lowered nor raised. The voluntary mechanism is therefore eliminated, but the bladder nerves are intact. Fluoroscopy shows that in such a case the patient cannot voluntarily start or stop micturition. What happens is that "automatic micturition" is set up. The entire act is taken by the detrusor muscle which contracts spontaneously once it is stretched to a certain point. Urination must then occur, and is started and stopped independently of the will. It is pointed out that this was a surprising discovery, since in the past we have been led to believe that bladder automaticity developed only after transection of the cord. In a case of *tabes dorsalis,* with elimination of the voluntary mechanism, the same type of automatic bladder was set up.

Pyelography After Uretero-Intestinal Anastomosis.

M. SCHNITTMAN (*The Journal of Urology,* September, 1948) states that with improved operative technique the percentage of normal pyelograms after uretero-intestinal anastomosis has been increased from 25% to 80%. The following principles are considered responsible for this improvement. (i) Mobility of the bowel in the recto-sigmoid region is ensured. This part of the bowel is mobilized freely if it is not already very mobile. The bowel should be made to fit the course of the ureter rather than the reverse; this precaution is to prevent the ureter from taking an "uphill" course. (ii) A minimum of ureter is freed. In this way, angulation is avoided, and unnecessary injury to the blood supply of the duct is prevented. (iii) An adequate submucosal trough is formed. This is probably the most important step of all. The trough must accommodate the ureter very loosely, and so be able to accommodate early œdematous swelling, as well as to allow for subsequent fibrotic changes. This ideal is obtained by the construction of a very wide trough, each lateral flap being at least 1·5 centimetres wide. (iv) Avoidance of excessive or redundant length of ureter between its exit from the peritoneum posteriorly and its entrance into the trough is important, but at the same time tension must be absent. There were 21 separate transplantations in the series reported, these being in eleven patients. With 17 of the ureters normal pyelograms were obtained up to twelve months afterwards, with two the result was intermediate and with two it was poor.

British Medical Association News.

THE OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA (BRITISH MEDICAL ASSOCIATION).

THE ninth annual general meeting of the Ophthalmological Society of Australia (British Medical Association) was held at the Medical Society Hall, Albert Street, East Melbourne, on October 4, 5, 6 and 7, 1949, DR. A. H. JOYCE, the President, in the chair.

COUNCIL PROCEEDINGS.

The following office-bearers for the year 1949-1950 were elected at a meeting of the Council:

President: Dr. W. Lockhart Gibson.
Vice-President: Dr. J. Bruce Hamilton.
Honorary Treasurer: Dr. A. E. F. Chaffer.
Honorary Secretary: Dr. A. L. Lance.

The following State representatives were elected:

Queensland: Dr. F. Garrett Scoles.
New South Wales: Dr. D. A. Williams, Dr. A. E. F. Chaffer, Dr. A. L. Lance.
Victoria: Dr. F. G. Fenton, Dr. J. McBride White, Dr. R. F. Lowe.
Tasmania: Dr. J. G. Hamilton.
South Australia: Dr. G. H. Barham Black.
Western Australia: Dr. D. D. Paton.

Dr. J. H. Doggart and Dr. T. K. Lyle were elected honorary members.

SCIENTIFIC SESSION.

The scientific session of the annual meeting was formally opened by Dr. Victor Hurley, President of the Federal Council of the British Medical Association in Australia.

President's Address.

The President, A. H. Joyce (Melbourne), began his address by thanking Dr. Victor Hurley, the President of the Federal Council of the British Medical Association in Australia, for having come to open the meeting. He also welcomed the overseas and other visitors. The meeting was the ninth annual general meeting of the society and the occasion was the tenth anniversary of the inaugural meeting. After referring to the changes which had taken place during the previous ten years and particularly to the six anxious years of war, the President said that in regard to the teaching of ophthalmology Australia had had to rely almost entirely on her own resources. A diploma in ophthalmology had been introduced in the Universities of Melbourne and Sydney and the Royal Australasian College of Surgeons had a Fellowship in Ophthalmology. In spite of the time and energy given to courses of instruction, they could not claim that the instruction was all that they could wish it to be. It would be an advantage if closer coordination could be arranged between Sydney and Melbourne in regard to the standards required and the courses in general. In Melbourne the centre of teaching was the Eye and Ear Hospital, but some of the instruction, and an important part of it, should be given at a general hospital. Australia should also endeavour to establish institutes after the manner of those existing in England and other countries. Such institutes were needed because Australia was isolated in two ways—by distance from other countries, and by distance between its own centres of learning.

Dr. Joyce then referred to the keeping of records. He said that, even though clinicians might keep excellent individual histories, they kept no index of cases. He thought that it was not impossible to have a central register to which all difficult and unusual cases could be reported with details of the condition and of the treatment given. He had discovered that there were no statistics of eye cancer in Australia—the few figures which did exist were included in percentages with cancer of the nervous system. A *questionnaire* had been sent to every member of the society and a good response had followed. An analysis of the cases was being undertaken by Dr. Hugh Ryan and it was hoped that later on a cancer register for the eye would be established for the whole of Australia.

The President then passed to a subject in which he claimed a particular interest—the endeavour to save sight as well as life, and in particular to avoid the horror of removing an only eye from a patient who had the misfortune to have an intraocular tumour. He referred to the pioneer users of radiation for eye diseases. He then referred to the work of Stallard and Foster Moore, who had shown that retinoblastomata and, to a lesser extent, melanomata were radiosensitive, and also to that of Algernon Reese and Martin, who had used fractional irradiation with small skin portals. He mentioned some of the early work done in Melbourne and said that he with Dr. Archie Anderson and Dr. Kaye Scott had been working as a team. He described five cases in which they had undertaken treatment. He said that the results were encouraging. Three of the patients suffered from retinoblastoma (two of them had three tumours in one eye) and two from melanoma.

A Message from Sir John Parsons.

At the request of the President, DR. J. H. DOGGART read a message of goodwill to the society from Sir John Parsons, of London.

The Movement of Macrophages in Corneal Grafts.

PROFESSOR IDA MANN (Oxford) read a paper in which she described some preliminary observations on the movement of macrophages in corneal grafts. She said that the problem of the origin of the cells in a successful corneal graft was complicated. It was not known whether the continued transparency of the graft was associated with survival of the donor corneal corpuscles or with their replacement by host cells, or with both. In other words, was the graft a true graft or did the implanted collagenous stroma act as as inert scaffolding for the support of regenerating host cells? An experimental approach to the problem was possible since macrophages could be vitally stained and observed from day to day without its being necessary to kill the animal. The technique had been used in studies following chemical injury. It was known that single macrophages entered the normal cornea from the limbus from time to time and were motile within it, and also that in conditions of chemical injury which killed the corneal corpuscles, macrophages from the subconjunctival tissue entered the cornea in large numbers and took an active part in the regeneration of both the cellular elements and the damaged collagen fibrils. The macrophage reaction occurred readily in the absence of vascularization of the cornea, and there did not appear to be any definite relation between the stimulus to macrophage reaction and that to formation of new blood vessels. Professor Mann described the technique which she had used for the introduction of the blue stain, and also some preliminary experiments undertaken in order to discover whether the constituent steps in the graft operation had themselves any effect on the movement of macrophages. These steps included the insertion of a suture, cutting with a trephine but not perforating, removal of a small piece of *substantia propria* but not perforating, and perforating with a trephine without removal of the disk. It was found that after these procedures very little macrophage activity occurred and that what did occur was independent of œdema, vascularization or infection. After these preliminary operations homoplastic and autoplastic corneal grafts were made. A corneal trephine was used and the disk was inserted without the use of sutures or of a conjunctival flap. Professor Mann described in detail four successful grafts (two were autoplastic). In all four successful grafts macrophages entered the graft in numbers during the period when it was slightly swollen and cloudy. They could no longer be seen when the graft was clear; this did not mean that they were not present, because it was known from previous work that as they differentiated in healing avascular corneal injuries they lost their blue colour. Good results were obtained with both homoplastic and autoplastic grafts and in spite of the presence of synechiæ. The formation of a temporary synechia in the rabbit appeared to be beneficial, since it afforded a direct route for the entry of reparative cells. Professor Mann stated her belief that the experiments led to the tentative conclusion that the graft was not a true graft, but provided a non-cellular collagenous scaffolding within which some at least of the host cells could reestablish themselves.

Orthoptics.

T. KEITH LYLE (London) read a paper entitled "Whither Orthoptics?" He said that when orthoptic treatment first became popular in England in the early 1930's it was heralded by many as the long awaited answer to the problem of the treatment of strabismus in children. It had been argued that some forms of reeducative treatment by means of exercises was needed. The rationale of this treatment had been based on the theory that a defect of binocular function, namely, "a defect of the fusion faculty", was the essential cause of strabismus. It was now generally held that in most cases the defects of binocular function were largely the result of the deviation and not its cause. To the lay public there had been something novel and perhaps agreeably unorthodox about treatment by means of

exercises, although in the lay mind exercises suggested some sort of physical jerks rather than the somewhat complicated retino-cerebral stimulation which orthoptic treatment actually involved. The immediate reaction to any new form of treatment tended to be either over-enthusiastic or precisely the reverse. He, Dr. Lyle, had been one of the early enthusiasts for orthoptic treatment. In the days when he, with Miss Jackson, wrote a book on practical orthoptics it had been seriously thought that orthoptic treatment by itself could effect a cure in a large percentage of cases of strabismus and that it was justifiable to carry out such treatment over a period of many months or even years at an enormous expenditure of time and possibly of money in order to restore binocular single vision. Nowadays ophthalmologists had a different conception of the use of orthoptic treatment. They did not regard it essentially as a method of curing a squint, but rather as a method of stimulating and reeducating binocular vision. In most instances angles of manifest deviation that were not overcome by correction of refractive errors were far more rapidly and more efficiently dealt with by means of operation than by orthoptic treatment alone.

Orthoptic treatment consisted essentially of retino-cerebral stimulation of both eyes simultaneously along their visual axes. It aimed to make the two eyes work harmoniously together with the enjoyment of binocular single vision. It consisted of overcoming suppression of the image of one eye, of teaching fusion or more commonly of reeducating fusion and of developing stereoscopic vision. Some people had taught that in strabismus in children, in which correction of the refractive error had not overcome the deviation, orthoptic treatment could do no more than, or even not as much as, surgical treatment accurately performed so as to render the visual axes parallel, provided that the visual acuity of each eye was approximately equal or had been made so by appropriate occlusion treatment. Dr. Lyle said that he had to admit that he was in general agreement with this view, provided the patient was treated early and not neglected. The primary concern of orthoptics in the case of very young children was in the testing of visual acuity by the "E" method and the direction and supervision of occlusion in order to equalize the vision of the two eyes. At the same time orthoptics had an important place in the treatment of certain cases of strabismus, especially those in children old enough to cooperate for exercises on the synoptophore, although such treatment might be auxiliary to operation.

Dr. Lyle went on to explain that in order to present as true a picture as possible of the treatment of strabismus in children, he had looked up the notes of all his own patients whom he had treated during the previous three years. The only cases he had excluded were those in which the squint was associated with a high degree of amblyopia that could not be corrected, and in which operation was performed merely for cosmetic purposes. First there was a group of 21 cases in which the squint had been controlled by correction of the refractive error. Of the 21 patients nine acquired binocular single vision with or without glasses and without any orthoptic treatment; ten acquired binocular single vision with or without glasses as a result of orthoptic treatment (in addition to the correction of the refractive error); and two had binocular single vision with glasses, but not without. A second group comprised 50 cases of convergent strabismus in which operation was performed. These had been divided into three groups. The first group comprised 30 cases in which normal retinal correspondence had been present when the patients were first examined. Operation had been successful in 16 of these cases; in seven the success was doubtful, because, although the visual axes were parallel, fusional duction power was poor; in seven operation was unsuccessful. The second group among the 50 cases included 14 in which abnormal retinal correspondence was present when the patients were first examined. The third group comprised six children who were too young or insufficiently cooperative for accurate estimation of the binocular functions.

After pointing out that there were circumstances in which orthoptic treatment might be dangerous, Dr. Lyle discussed divergent strabismus and concluded that in the majority of cases operation was indicated. In regard to cases of heterophoria in which symptoms of decompensation were present, there was not the slightest doubt that, provided the diagnosis was correct, the results were good in almost 100% of cases. The group on which this conclusion was based included 67 patients. In his general conclusions the speaker included the statement that to attempt to diagnose and treat patients suffering from strabismus without the assistance of an orthoptic department was rather like trying to treat and diagnose disease of the chest or alimentary tract without the use of radiography.

Blind Eyes: Incidence, Causation and Prevention.

K. B. REDMOND (Orange) read a paper in which he discussed the incidence, causation and prevention of blind eyes. He defined a "blind eye" in terms of the definition of blindness adopted by the society as follows: "A blind eye exists when there is central visual acuity of $^{6}/_{60}$ or less with correcting glasses, or central visual acuity of more than $^{6}/_{60}$ if there is a field defect in which the peripheral field has contracted to such an extent that the widest diameter of visual field subtends an angle of no greater than 20 degrees." The paper was based on 700 cases of unilateral blindness and 115 cases of bilateral blindness discovered by the examination of the records of 10,000 patients consulting an ophthalmic surgeon over a period of ten years; no selection had been used.

The causes of bilateral blindness included: infections and toxæmias; senile macular lesions; cataract; glaucoma; retinal vascular anomalies; defects of congenital, hereditary and developmental origin; myopia; optic atrophy from cerebral tumour; injury; detachment of retina; and hysterical amaurosis. Of the 115 patients 79 were fifty years of age or over; 24 fell in the age group of fifteen to forty-nine years, and 12 were under fifteen years of age. The pathological causes of unilateral blindness included: cataract (133); injuries (120); infections and toxæmias (102); vascular disorders (74); glaucoma (57); amblyopia (53); senile macular lesions (48); defects of congenital, hereditary and developmental origin (48); myopia (34); retinal detachment (22); optic atrophy (5); melanotic sarcoma (2); hysteria (2). In 404 cases the patients were over fifty years of age; 150 patients were in the age group fifteen to forty-nine years; and 146 were younger than fifteen years. The principal causes of blindness among patients under fifteen years of age were injuries, amblyopia, and congenital, hereditary and developmental anomalies; in those aged fifteen to twenty-nine years the chief causes were injuries, amblyopia, myopia and infections; in those from thirty to forty-nine years of age the chief causes were infections, injuries and myopia; in those from fifty to sixty-nine years of age the chief causes were infections, cataract, vascular disorders and glaucoma; and in those seventy years of age and over, cataract, senile macular lesions, vascular disorders and glaucoma.

Dr. Redmond pointed out that if the causes operative in the different age groups were evaluated in terms of expectation of life, cataract, retinal vascular disorders, glaucoma and senile macular lesions had an exaggerated significance; the prominent conditions were injuries and these were followed by amblyopia, defects of congenital, hereditary and developmental origin, infections, toxæmias and myopia.

In regard to the social, economic and professional causes of blind eyes, Dr. Redmond grouped the 700 cases under the following headings: treatment inadvisable, 80 cases; blindness inevitable, 395 cases; blindness due to inefficient social services, 18 cases; blindness due to neglect of patient or parent, 97 cases; blindness due to neglect of general practitioner, 12 cases; blindness due to neglect of oculist, 13 cases; cases in which patient was still under active treatment, 85.

In his discussion on the prevention of blind eyes, Dr. Redmond laid stress on the need for cooperation of oculist, general practitioner, social worker, public and patient; he made a special plea for the education of public and patient.

Tumours of the Iris.

KEVIN O'DAY (Melbourne) read a paper on tumours of the iris. One epithelial inplantation cyst and six tumours involving the iris were described. The first tumour was a metastasis from a chorioidal melanoma; the second was a diktyoma of the ciliary body invading the iris;; the next three were infiltrating tumours of the iris, the last of which had a clinical history of six years' duration; and the last was a leiomyoma. Dr. O'Day demonstrated the deceptive nature of iris tumours, in particular of the infiltrating tumours, and he emphasized the necessity for careful examination of blind painful eyes which had come to enucleation. The paper, which was accompanied by nineteen photomicrographs, concluded with a discussion on unpigmented tumours. Dr. O'Day said that these were probably never the so-called leucosarcomata, but leiomyomata.

Corneal Dystrophy.

J. H. DOGGART (London) began his paper on corneal dystrophies by quoting a remark made twenty years previously by Sir John Parsons that "we know nothing about the corneal dystrophies". That might be repeated with equal truth at the present time. Parsons had intended to emphasize the fact that no "luminous interpretation" of corneal

dystrophies had emerged from a "welter of laborious detail". The word dystrophy implied interference with the welfare of a tissue and did not put the responsibility upon any particular agent. If it was stated that such-and-such a condition was a corneal dystrophy, most ophthalmologists would take it that the clinical phenomena were not satisfactorily explained by influences known to be deleterious towards the average cornea. The term suggested that chronic degenerative changes were envisaged. Violent outbursts of idiopathic keratitis would be more likely to be designated allergic. In the course of routine examination the cornea was scrutinized, whether the symptoms pointed to any corneal lesion or not, and it was necessary to be on the alert for any abnormal appearances. These might direct attention to other parts of the eye or to remote parts of the body. In a large proportion of cases the conclusion would be reached that the corneal changes were attributable to a noxious agent. But sometimes cases were seen in which careful clinical and laboratory search failed to supply any clue to the causation. In such circumstances it was convenient to speak of dystrophies, provided it was realized that the application of this tag did not constitute new knowledge, any more than the title "idiopathic" purported to be interpretative. As a result of experience corneal dystrophies had been tidied up into a number of pigeon holes; new types were becoming progressively improbable, though not impossible.

In discussing the ætiological factors of corneal disturbance, Dr. Doggart referred to the conception of a defect in the germ plasm as responsible in certain cases. The suggestion was that tissues were endowed with properties foreshadowing early decay. This did not serve to explain the corneal dystrophies, because their characteristic signs did not resemble the changes wrought by time. For example, Fuchs's dystrophy could not be simply discussed as premature senility. At the same time the existence of familial keratitis strikingly illustrated the truth that corneæ destined to show pathological changes in the second decade of life could be free from abnormal signs meanwhile. It would not be easy to account for the dystrophies by postulating damage from intrauterine malnutrition and maternal toxins, even though rubella had been transformed from a harmless nonentity into a mutilating monster. Such influences could bring about congenital opacity and other forms of corneal maldevelopment, but they could not easily be linked up with Fuchs's epithelial dystrophy or band-shaped opacity. Birth injury might cause congenital abnormality, but in such cases there was no clear connexion with the dystrophies. After stating that age and sex were clearly important as ætiological factors, Dr. Doggart mentioned the following postnatal influences:

(i) Insufficient protection by the eyelids because of cicatricial contraction, facial palsy or proptosis.

(ii) Disease of the lachrymal and mucous glands. In this connexion Sjøgren's disease or *kerato-conjunctivitis sicca* was mentioned.

(iii) Metabolic disorder, endocrine derangement and nutritional defects, as prime causes and also as aggravating factors, in which role they often overlapped. Examples had been recorded in myxœdema and in riboflavin deficiency. Ferraro and his co-workers also had described epithelial disorganization in the corneæ of rats fed on a diet deficient in valine.

(iv) Neurological influences. The fact that epithelial œdema was a salient feature in many types of corneal dystrophy had encouraged some authorities to suppose that the corneal dystrophies were chiefly neuropathic in origin.

(v) Physical influences—heat, dust and cold.

(vi) Chemical factors.

(vii) Aphakia. Aphakic disintegration of the corneal epithelium tended to be progressive and was a source of great disappointment, because the interpalpebral region suffered most, so that visual acuity failed.

(viii) Infection.

Dr. Doggart referred to clinical signs which were often present in the dystrophies, though as a rule they were not separately pathognomonic of the condition. Those mentioned included: epithelial œdema; calcification; fatty change; crystalline deposits; opacity of the corneal substance, which might occupy any layer and might consist of dots, streaks and irregular figures showing infinite variety of disposition; changes in Descemet's membrane; Descemet's endothelium; pigmentation; the presence of new vessels.

In conclusion Dr. Doggart dealt in some detail with Fuchs's epithelial dystrophy and marginal degeneration of the cornea. He pointed out that though the former condition was not common, failure to recognize it often led to disaster. When this happened patients were subjected to drainage operations in the erroneous belief that they were suffering from glaucoma. He discussed the incidence and differential diagnosis of both conditions and referred to the prognosis and treatment. His concluding statement was that, even if it was still true, as Sir John Parsons had declared, that nothing was known about the corneal dystrophies, they ought still to be kept on the list of nodding acquaintances.

"Dry Eyes" and Their Relation to Delayed Recurrent Mustard Gas Keratitis.

J. BRUCE HAMILTON (Hobart) read a paper entitled "A Note on Dry Eyes and Their Relation to Delayed Recurrent Mustard Gas Keratitis". He referred to a monograph published in 1948 by Professor Ida Mann with A. Pirie and B. D. Pullinger entitled "An Experimental and Clinical Study of the Reaction of the Anterior Segment of the Eye to Chemical Injury, with Special Reference to Chemical Warfare Agents". This monograph reported researches carried out on behalf of the British Ministry of Supply during the recent war. Dr. Hamilton said that when he decided to write a paper on delayed manifestations of mustard gas keratitis he had naturally turned to Arnold Lawson's work on the blind at Saint Dunstan's after the first World War. This was written in 1922 and contained no reference to mustard gas as a cause either of ocular irritation or of subsequent blindness. When Professor Mann's chart was consulted it was seen that the peak years for delayed mustard gas keratitis were 1933 and 1936—this was a period of more than fifteen years after the original injury. Dr. Hamilton asked whether it was possible to offer any explanation for such a long quiescent period other than a gradual shrinking of lachrymal and conjunctival secretions culminating in desiccation. In her monograph Professor Mann had referred to the protection afforded by the use of contact lenses and had stated that this was probably explained by the protection of the partially insensitive cornea from multiple minute injuries. Professor Mann had shown that mustard gas and collagen formed a compound in the cornea which contained much sulphur. This compound incited the disposal in the cornea of cholesterin and fat which in themselves acted as irritants and provoked a secondary breakdown of the scar at a later date. Dr. Hamilton asked whether the corneal scar broke down *per se*, or whether the breakdown was due to receiving multiple injuries as suggested by Professor Mann. He referred to a paper published by himself in 1947 in which he expressed the view that mustard gas ultimately caused an essential shrinkage of the conjunctiva and the lachrymal gland, depending on the severity of the original trauma—this essential shrinkage produced a dry conjunctival sac and desiccation of the cornea followed by recurrent ulceration. He suggested that at least one of the "minute injuries" to which the insensitive mustard gas cornea was subjected was desiccation. He appealed to all who had under their care patients with this kind of condition to estimate the tear secretion by Schirmer's test. He illustrated his remarks by reference to five cases.

Cataract Development.

RONALD F. LOWE (Melbourne) read a paper entitled "Some Thoughts on Cataract Development". He reviewed the nomenclature of the zones of optical discontinuity as revealed by the slit-lamp and compared the terms used by different authors. He suggested that the following terms should be used: embryonic nucleus (to include the central dark interval and the plano-convex areas), fœtal nucleus (the zone containing the Y-sutures surrounding the plano-convex areas), adult nucleus and cortex. He said that the cause of familial cataracts might be the failure of abnormal genes to produce specific enzymes for the completion of some phases of intermediary metabolism of the lens. Certain layers of the lens were more prone to be affected than others, and susceptibility increased when new phases of growth were initiated. Lamellar cataracts were therefore commonly seen either encircling the embryonic nucleus or situated deeply within the adult nucleus. The former period corresponded to the development of the earliest secondary lens fibres, and the latter to the beginning of an independent existence by the child. Illness or environmental deficiencies, by affecting the lens fibres at an appropriate period of life, might result in opacities similar to those genetically determined. Dr. Lowe showed examples of different lamellar cataracts to illustrate these possibilities.

The Treatment of Acquired Ocular Palsy.

T. KEITH LYLE (London) read a paper on some aspects of the treatment of acquired ocular palsy. He referred to the view formerly held that cases of acquired ocular palsy, which were not due to active organic disease, and in which spon-

taneous recovery did not occur, were regarded as incurable. This was not true. Experience in the recent war had shown that in many cases of traumatic ocular palsy in which spontaneous recovery had not occurred, diplopia could be completely overcome by surgical adjustment of the extrinsic ocular muscles. Moreover, surgical treatment could be used in other conditions, not of traumatic origin, in which spontaneous recovery of the affected muscle or muscles or of their nerve supply had not occurred. Before operation was considered in any case of ocular palsy, it was essential to make quite certain that (*a*) no further spontaneous improvement was likely to occur, (*b*) no active organic disease was present which might lead to further change in the ocular deviation. Dr. Lyle then discussed the forms of ocular palsy suitable for surgical treatment under two headings: (*a*) ocular palsy of traumatic origin; (*b*) ocular palsy due to organic disease. The former he divided into palsy due to a nerve lesion, relative ocular palsy due to orbital trauma, direct trauma to a muscle. The second group of ocular palsies due to organic disease he divided into those due to exophthalmic ophthalmoplegia and those of indeterminate pathology. He dealt with each division in turn and described several cases in detail in each.

The Cutler Implant.

J. L. R. CARTER (Launceston) showed a motion picture of the Cutler implant—its insertion and the end result with the patient wearing an artificial eye.

Ophthalmic Research in London.

PROFESSOR IDA MANN (London) gave a short account of the general set-up for ophthalmic research in London. She explained that when the national health service under the control of the Government was imminent it had been realized that special arrangements would have to be made to secure the continuance of research. There had been in London three ophthalmic hospitals—Moorfields, Westminster and the Central London Hospitals. Wards for the treatment of patients had been concentrated at the first two institutions and the Central London Hospital was transformed into an ophthalmological institute. Here research was conducted and clinical investigations were carried out for clinicians in the other two institutions. Here also lectures were given to post-graduate students. The institute was a member of the Federation of Post-Graduate Institutes of the University of London and was under its ægis. The research was sustained by the University of London and by grants to individual workers from the Medical Research Council. The post-graduate students did their clinical work at Moorfields and at the Westminster Hospital. Professor Mann's talk was illustrated by a series of lantern slides.

Ophthalmology in Great Britain under the National Health Service.

FRANK W. LAW (London) had been asked to send a paper on the practice of ophthalmology in Great Britain under the National Health Service. This he did and the paper was read by J. H. Doggart. He pointed out that the medical profession was irrevocably committed to service under the Act and could not honourably withdraw. What it could do was to take a firm stand on the interpretation of the Act and demand modification of its provisions where they seemed unacceptable. Before the health service was initiated it had been recognized by a joint committee of ophthalmic surgeons, opticians and administrators that it would be impossible to launch a full refraction service manned by medical men. It was therefore decided to institute the Supplementary Ophthalmic Service which was to cover the provision of refraction services both by medical men and women and by opticians. At length a scheme was devised which appeared reasonable and convenient to all parties as a temporary measure. It was important to note that a large number of participants were willing and anxious that the temporary scheme should continue indefinitely, but that it possessed many features whose perpetuation seemed undesirable to others. Before a member of the public made use of the Supplementary Ophthalmic Service, he had to obtain a certificate from his general practitioner to enable him to do so; subsequently he might go without a passport to the ophthalmologist or optician of his choice. The patient with a certificate went to a doctor or an optician and then to a dispensing optician of his choice who would supply glasses. An optician might act both as a refractionist and as a dispensing optician. For the refraction the ophthalmic practitioner received at first a fee of £1 11s. 6d. on the basis of two an hour, but latterly the fee had been reduced to £1 5s. on the basis of five in two hours. The optician received 15s. per refraction; in addition he received a fee of £1 5s. per pair for measuring the patient for glasses when they arrived and was reimbursed by the Government for the total cost of the glasses supplied.

Another point which had given rise to considerable discussion was the demand by medical negotiators that any patient seen by an optician who failed to read $^6/_6$ with either eye should be referred to a medical man. On the grounds that the optician was a technician and not a medical man, the decision in question seemed justified. The opticians, however, resented the provision as a slur on their capacity and had stated that it was administratively impossible since 70% to 80% of patients failed to achieve $^6/_6$ vision and reference of such numbers was impossible. Dr. Law was unable to accept the figure; he added that the higher it was, the more strongly it might be held to provide an argument against the excellence of a government-sponsored system, which placed its citizens in the hands of practitioners who did not pretend to be qualified to deal with conditions presented by such a proportion of them.

The Supplementary Ophthalmic Service was to last until the hospital system was ready to receive and to deal with the vast mass of refraction work presented. For this decentralization would be essential. One-half of the problem would be the provision of sufficient ophthalmic surgeons to man such a service and the other half would be the provision of health centres or other buildings in which the work could be done. The present cry of the out-patient staff in ophthalmic hospitals was: "Let us do half the work twice as well."

The absorption of the Supplementary Ophthalmic Service into the Hospital Service, with its consequent elimination as an entity, was to take place gradually area by area as soon as local conditions were ready for it. It was intended that opticians should work in the hospital and clinic refraction departments with, and under the supervision of, ophthalmic surgeons—another aspect of the scheme which raised acute problems.

Dr. Law also made reference to the effects of the health service and to the enormous number of spectacles required; he referred also to the varying effect on private practices. He concluded by expressing the opinion that the old confidential doctor-patient relationship could not survive the present-day changes.

Central Serous Retinopathy.

J. H. DOGGART (London) read a paper dealing with central serous retinopathy. He explained that few people outside Japan had seen many examples of the condition. He, himself, had seen only 17, but in Japan records of 600 cases had been assembled in five years. It was a condition to which a variety of names had been given, including preretinal œdema, central annular retinitis, *chorioretinitis centralis serosa*, idiopathic flat detachment of the retina, *retinitis centralis angioneurotica*, and central angiospastic retinopathy. Of Dr. Doggart's 17 patients, 14 were women; but in Kitahara's series of 150 cases males were affected twice as commonly as females. The onset usually occurred between the ages of twenty-five and forty years. The Japanese ascribed importance to a number of ætiological factors such as tuberculosis, syphilis, blood-borne toxins and infection of the nasal sinuses in association with undue sensitivity to light. Many other causes had been suggested and the main rivals in causation were infection and vascular changes; some observers admitted that both might be operative. If vascular changes were responsible there were many different agents which might set the changes in motion.

The first symptom was blurring of central vision. Sometimes deranged colour perception was noted; colours looked dirty. Positive scotoma was a prominent feature in the active stage. One observer held that disappearance of the positive scotoma was the most important subjective signal that œdema was on the ebb.

During the stage of active œdema the chief ophthalmoscopic change was an oval or circular blister at the posterior pole of the eye. Occasionally clusters of small gleaming dots appeared in the immediate neighbourhood of the blister, but more noteworthy was its curious mottling by deep, greyish-yellow, ill-defined dots varying in size. Macular hæmorrhages sometimes appeared. If the œdematous region was relatively small and if resolution soon supervened, then the disease might clear without any residual signs. In a large proportion of cases, however, the disturbed area was permanently marked by pigmentary degeneration. In favourable cases the fluid absorbed within a few weeks, but the rate of resolution varied considerably, so that years might elapse before any flattening was manifest. The condition might be unilateral and recurrence was far from being a negligible possibility.

After giving details of several cases, Dr. Doggart discussed the differential diagnosis and said that among the explanations attached to patients' symptoms at various times were retrobulbar neuritis, tobacco amblyopia, anxiety neurosis, malingering and hysteria. So long as the observer had satisfied himself that a macular lesion was present, he was not likely to mistake the active stage of central serous retinopathy for anything else. At the same time there were cases in which the macular signs might equally well be explained by a former attack of the disease or by some very different condition.

In regard to the pathology it was generally agreed that there must be an increase in the permeability of the macular capillaries, and especially of those belonging to the chorioidal network, which was exclusively responsible for nourishing the juxta-foveal region. Such increased permeability could be due to toxins produced locally or conveyed to the site from remote foci of infection. Some of the changes could readily be fitted into the framework of allergic theory; one observer had claimed that in two cases vaccination acted as a trigger for the allergic explosion.

In the matter of treatment judgement would have to be reserved until a long list of successes had been scored by more than one observer. In prognosis undue optimism prevailed, but many victims sustained lasting impairment of sight in one or both eyes.

Dr. Doggart concluded by remarking that central serous retinopathy was not a common disease, but should always be borne in mind as a possible explanation for blurred central vision suddenly afflicting an otherwise healthy person in youth or early middle age.

Gonioscopy in Glaucoma.

HUGH RYAN (Melbourne) read a paper in which he set out to indicate the value of gonioscopy as a routine measure in the examination of patients with glaucoma and to emphasize the particular value of that investigation in pre-operative, operative and post-operative treatment. In his experience it had been found of great help in deciding what operation was best, and, in unsuccessful cases, in finding where the operative technique might have failed and the factors which predisposed to success or failure. Dr. Ryan traced the development of gonioscopy from the work of Trantas in 1907 up till 1936, when the instrument used had become cumbersome and expensive and the use of gonioscopy languished. In 1938 matters had improved because Goldman in that year devised for gonioscopy a plastic contact lens with a built-in mirror for use with an ordinary slit lamp. In 1945 Allen and O'Brien had improved on the Goldman lens by using a plastic prism instead of a mirror to illuminate the angle—the slit lamp was still used. Dr. Ryan described the use of the gonioscope and illustrated the normal appearances of the angle and its relationship to normal anatomical structures. He said that the most important landmark was Schwalbe's line which represented the end of Descemet's membrane. Since it was covered with pigment granules, it was as a rule easily found. Inability to find it suggested that the angle was closed or abnormal. Dr. Ryan mentioned circumstances which made it difficult to tell whether the angle was closed or open. He then described his studies of 40 glaucomatous eyes which had been subjected to various surgical operations. In his discussion on iridectomy he concluded that this operation acted by reopening the blocked angle rather than by establishing a passage away from the posterior to the anterior chamber; he also discussed the blocking of a trephine hole. He showed by examples that gonioscopic findings might explain the success or failure of operation, and ended with a plea that use of the gonioscope should be universally adopted as a routine procedure in cases of glaucoma.

A Glaucoma Clinic.

N. M. MACINDOE (Sydney), on behalf of himself and D. SHORTRIDGE (Sydney), read a paper entitled "First Impressions of a Glaucoma Clinic". He said that early in the previous year a glaucoma clinic had been started at the Royal Prince Alfred Hospital, Sydney, and had been conducted since then on the first Tuesday in each month. Patients were referred to the clinic by the out-door ophthalmic surgeons for either investigation or follow-up. A register was kept, with a special sheet on which all relevant data were recorded. Patients were given a form telling them the date of their next appointment and reminding them of the serious nature of their disease. If a patient failed to attend on the appointed day, a duplicate of the slip was sent him by post, but this was rarely necessary. Investigation and treatment were along routine lines and the patient was referred back to his surgeon from time to time. Field studies were attempted in every case and serial observations on intraocular pressure as measured by tonometry were recorded. Readings were taken before and after mydriasis in doubtful cases and the appearance of the optic disk, the visual acuity and the history were all considered. Dr. Macindoe and his co-author compared glaucoma to diabetes and stated that special clinics in the latter condition had proved their value. They made the patient realize the importance of his malady and its treatment and acted as a barrier to his drift from treatment. It was not suggested that only one doctor should treat glaucoma in a hospital, but simply that the hack work associated with it should be segregated in the interests of more efficient treatment.

Retinitis Punctata Albescens.

GEORGE A. BREW (Melbourne) read a paper in which he reported two cases of familial night blindness with field loss and early cataract; fundal changes were present suggestive of *retinitis punctata albescens*. Appleman had given a concise definition of that condition as follows: "A stationary night blindness dating from early life, probably congenital, with a multitude of white spots in the fundus, not accompanied by any pigmentary changes or progressive loss of vision as occurs in *retinitis pigmentosa*." Dr. Brew described the findings in detail and said that the retinal appearances in the first case were probably unique. One of his objects in reporting the cases was to demonstrate that complete investigation of such conditions and of the allied and better known *retinitis pigmentosa* demanded careful perimetry.

"A Cross Section of Crossed-Eyes."

J. RINGLAND ANDERSON (Melbourne) read a paper in which he analysed over 300 cases of convergent strabismus. He drew attention to the influence of inheritance, birth injury and infection and said that the age of onset and the age at which treatment was commenced were very important. Amblyopia was the most serious obstacle to cure; it was much too common and the education of both parents and doctors in regard to it was necessary. Anomalous inter-retinal correspondence was another obstacle. As a rule it could be converted into the normal state, though age of onset and the presence of a vertical deviation made this more difficult. Early and alternate occlusion was recommended as the most valuable treatment. Early operation and thorough orthoptic training were advocated. Dr. Anderson said that the condition should be cured before school age. He concluded by insisting that team work was essential in the treatment—"the bowlers should know the wicket keeper".

Post-Rubella Retinal Conditions in Western Australian Children.

C. MORLET (Perth), in presenting a short report on post-rubellar retinal conditions in Western Australian children, said that he had hoped to make his report at the 1948 meeting at Perth, but had been unable to attend owing to illness. He gave a short summary of the results of his survey. The population of Western Australia was 480,000 and nearly 300,000 people lived in the metropolitan area. In the State he had discovered 82 so-called rubella children—those whose mothers had suffered from rubella in the early stages of pregnancy. All the 82 children were deaf with two exceptions; none were completely blind. Of the 82 children he had been able to examine only 60. Ten were in the Education Department at Boulder. The Government did not see its way clear to send the children to him for examination and he could not undertake the 700 mile return journey to examine them at his own expense. Of the 72 children in the metropolitan area, eight had so far evaded him by absence from school and four were private patients of his colleagues. Of the 60 children examined, 52 had $^{6}/_{6}$ vision in each eye, but in 36 unmistakable chorioido-retinal changes were present. In other words 60% of "rubella children" examined in Western Australia had abnormal ocular fundi. It was interesting that in no single case so far had loss of visual acuity attributable to retinal changes been found. Dr. Morlet suggested that a survey should be made in each State and the visual acuity of the children periodically checked.

ANNUAL DINNER.

The annual dinner was held at Menzies Hotel and about 80 members were present. There were four guests—Professor Ida Mann, Dr. J. H. Doggart, Dr. T. K. Lyle and the Editor of THE MEDICAL JOURNAL OF AUSTRALIA. The toast of the

society was proposed in felicitous terms by Dr. Cedric Cohen and Dr. J. Bruce replied. The toast of the visitors was proposed in characteristic fashion by Dr. Archie Anderson and Dr. Darcy Williams and each of the visitors replied.

TIME AND PLACE OF THE NEXT ANNUAL MEETING.

It was noted that the next annual meeting would be held at Brisbane in May, 1950, concurrently with the Seventh Session of the Australasian Medical Congress (British Medical Association).

Special Abstract.

PENICILLIN DOSAGE FORMS FOR SYSTEMIC INFECTIONS.

PENICILLIN has become established as one of the major therapeutic agents at our disposal. Many advances in its use have been made since it was first introduced. A useful survey of some of the current views concerning penicillin treatment with different dosage forms has been recently made by Chester S. Keefer.[1] The following summary of his paper should be of interest and practical value.

Parenteral Administration of Penicillin.

The most common forms in which penicillin is administered parenterally are as follows: (i) sodium or potassium salts of penicillin G for injection in aqueous solution; (ii) procaine penicillin G suspended in oil, water or oil and aluminium monostearate; (iii) combinations of crystalline sodium or potassium and procaine penicillin G for dispensing in an aqueous medium.

From the beginning of penicillin therapy the water-soluble salts have been widely used, being injected either continuously by the intravenous or intramuscular route, or, more commonly, intermittently by the intramuscular route. Treatment by means of intermittent injections is classed either as continuous or as discontinuous. With continuous treatment an attempt is made to inject a sufficient amount at regular intervals so that penicillin is present in the blood and tissues at all times during the twenty-four hour period. With discontinuous therapy the injections are spaced in such a way that penicillin is not detected in the blood and tissues at all times during the twenty-four hour period. The goal of treatment should be to effect recovery from infection in the shortest period of time with the least amount of inconvenience to the patient. Every infection presents an individual problem; the amount of penicillin required in the twenty-four hour period and the frequency of injections must be determined. The minimum effective dose has never been established for all infections; but it can be said that in the treatment of common infections such as pneumococcal pneumonia, hæmolytic streptococcal infections and gonococcal infections, the general trend in treatment with water-soluble salts has been to increase the amount of penicillin that is given at each injection, and to increase the interval between injections. For example, in the treatment of lobar pneumonia it has been found that the injection of 200,000 or 300,000 units twice a day is as effective as the injection of 25,000 units every three or four hours. Thus the trend has been towards discontinuous therapy when the water-soluble salts are employed; in this case the concentration of penicillin in the plasma and in the tissues is optimal during the first hours after injection, and then declines rapidly over a period of four to six hours. It has been recognized since the beginning of the use of penicillin that it is unnecessary to maintain a concentration of penicillin in the blood and tissues throughout the day in order to obtain optimal therapeutic results. The decision as to the total dosage in twenty-four hours and the frequency of injection will depend upon the type of infection that is being treated and the patient's response to therapy.

Indications for Use of Crystalline Penicillin G.

Crystalline penicillin G must be used for all oral therapy, for preparation of all solutions for topical application, such as intrathecal, intrapleural, intraabdominal, intraarticular and intratracheal, and in the preparation of aerosol solutions. It should also be used parenterally in combination with procaine penicillin G in serious infections, so that high plasma concentrations of penicillin may be obtained promptly. These so-called "booster" doses of penicillin may be repeated twice daily when the organisms are relatively resistant or when the infection is located in an area of the body where penicillin diffuses with difficulty.

Procaine Penicillin G.

Procaine penicillin G, a salt of penicillin that is relatively insoluble in water, can be suspended in sesame oil or in water. When 300,000 units of procaine penicillin suspended in one millilitre of oil or water are injected into the muscles, the penicillin is slowly released from the deposit. The plasma concentration of penicillin reaches its maximum within one to two hours, then decreases slowly. This contrasts with sodium penicillin G in aqueous solution, which produces a maximum plasma concentration within ten to fifteen minutes after injection, but the penicillin disappears from the circulating blood at a much more rapid rate. The maximum plasma concentration obtained with water-soluble sodium penicillin G is always greater than that observed after the injection of a similar amount of procaine penicillin G. In an attempt to produce an initially high plasma concentration of penicillin during the first thirty to sixty minutes, crystalline sodium or potassium penicillin G has been combined with procaine penicillin G so that in one millilitre of the aqueous material 100,000 units of the crystalline soluble salt are combined with 300,000 units of the procaine salt.

When procaine penicillin G is suspended in oil and aluminium monostearate is added, the penicillin is absorbed more slowly and more uniformly. The initial and peak plasma concentrations of penicillin are lower than when other preparations are used, but desirable plasma concentrations are present for a longer time. All the studies with this preparation show that it is exceedingly difficult to obtain a high plasma concentration, but that the level obtained can be greatly prolonged. It can be said that after the injection of 300,000 units of procaine penicillin G in water or oil, this amount is available to the tissues for a period of twenty-four hours, and in a concentration that is usually considered to be adequate for the control of the vast majority of penicillin-sensitive infections. After the injection of 300,000 units of the same salt combined with aluminium monostearate, this amount of penicillin is available to the tissues over a period of ninety-six hours, but the concentration of penicillin is always lower during the first twenty-four hours of treatment than when no aluminium monostearate is used.

Indications and Dosage.

A single daily injection of procaine penicillin G in oil or water is adequate for the effective treatment of most infections requiring penicillin. Larger doses—that is, 600,000 units once or twice a day—should be given for infections caused by organisms that are only moderately sensitive to penicillin, such as the staphylococcus and *Streptococcus viridans*. When procaine penicillin G in oil and aluminium monostearate is employed, the injections may be spaced several days apart. It has been reported that after one injection of this preparation containing 3,000,000 units, a plasma concentration of 0·1 unit per millilitre can be maintained for a week.

Dosage schedules of procaine penicillin G that have been used with success are as follows: pneumococcal pneumonia, 300,000 units given once a day for five to seven days; gonorrhœa, 300,000 units, one injection; subacute bacterial endocarditis, 300,000 to 600,000 units given once or twice daily for six to eight weeks; streptococcal infections of the throat, 300,000 units given once a day for five to seven days; staphylococcal infections, 300,000 to 600,000 units given once or twice a day for seven to fourteen days; prophylactic use, 300,000 units given once a day; post-operatively, 300,000 units given once a day; tooth extractions, 300,000 units given once, one hour before the extraction; puerperium, 300,000 units given daily for five days; tonsillectomy, 300,000 units given once.

Oral Administration of Penicillin.

Penicillin is available for oral administration in the form of either the sodium or the potassium salt of crystalline penicillin. It is not necessary to use a buffered preparation in order to obtain a satisfactory therapeutic result. The oral route is the logical way to give any potent drug, provided it is absorbed from the gastro-intestinal tract. It is convenient, it decreases nursing care and discomfort for the patient, it saves the time of the doctor and the nurse, and it should cost the patient no more. It has been demonstrated repeatedly that with penicillin the oral route is as effective as the parenteral route when adequate doses are used, and in addition produces fewer hypersensitive reactions.

[1] *The American Journal of Medicine*, August, 1949.

Dosage schedules that have been effective in the treatment of the various diseases have varied greatly. In general it can be said that three to five times the minimum effective parenteral dose of penicillin has been used with a favourable result. The dosage may be summed up as follows: pneumococcal pneumonia, 600,000 to 1,000,000 units given daily (150,000 to 200,000 units given daily for infants and children); gonorrhœa, 200,000 to 500,000 units for one day; hæmolytic streptococcal infection, 450,000 to 1,000,000 units given daily for five to seven days; staphylococcal infections of the skin, 1,000,000 units given daily for five days; Vincent's stomatitis, 400,000 to 600,000 units given daily for three to four days.

Carinamide.

Carinamide has been used as an adjunct to penicillin therapy to delay the excretion of penicillin by the kidney. It has been most useful with infections due to highly resistant organisms such as are seen in occasional cases of subacute bacterial endocarditis or staphylococcal sepsis. Thus, after a single dose of 500,000 units of penicillin given intramuscularly and three grammes of carinamide given orally, plasma concentrations varying from 28 to 34 units per millilitre can be maintained for a three-hour period.

Inhalation of Crystalline Penicillin G.

Methods have been developed for inhalation of penicillin dust directly into the respiratory passages, and have been used most extensively in the treatment of patients with bronchiectasis and other chronic infections of the respiratory tract as well as infections of the upper part of the respiratory tract. After the inhalation of 100,000 units of penicillin dust, there is good evidence that penicillin is absorbed. Maximum levels of penicillin in the blood plasma are obtained one hour after the inhalation, and penicillin can be detected for three to five hours after inhalation. A decrease occurs in the bacterial population of the nose, throat and sputum, the Gram-positive bacteria being particularly affected. Hypersensitive reactions in the mouth and throat or at the point where penicillin comes in contact with the skin have been reported in from 3% to 6% of patients. Systemic reactions are infrequent. This form of penicillin therapy is easily available for use in consulting room and home practice. It is usually recommended that at least one to three inhalations of 100,000 units of penicillin dust be used daily.

Special Correspondence.

NEW ZEALAND LETTER.

FROM OUR SPECIAL CORRESPONDENT.

Appointment of New Director-General of Health.

THE impending retirement on superannuation of Dr. T. R. Ritchie, who has been Director-General of Health for three years in succession to Dr. M. H. Watt, has led to the designation as his successor of Dr. John Cairney by the Public Service Commission. The appointment is subject to the usual Public Service Appeals. The Commission was advised by a special committee consisting of Sir Charles Hercus, Dr. E. H. M. Luke, Mr. Douglas Robb and Mr. P. E. Stainton.

John Cairney, D.Sc., M.D., F.R.A.C.S., who is fifty-one years of age, is at present Medical-Superintendent-in-Chief to the Wellington Hospital Board, in which service he has been since 1936. Prior to that he was Medical Superintendent of Hawera Hospital, and before that demonstrator, lecturer, and Associate Professor of Anatomy in the Otago Medical School for seven years. He thus approaches his new task with full experience—and a great reputation—in the hospital field. This is recognized as at once the largest and most attention-requiring field within the department's duties. For years the machinery has been creaking, and coordination between the department and the local boards has been inadequate.

Dr. Cairney faces a formidable task. Not only the hospital system, but the hygiene work throughout the country needs improved conditions and organization. In addition there has never yet been set up a structure to handle the numerous and important matters arising out of social security legislation in New Zealand—now nigh ten years old. His plans, therefore, will be followed with interest and hope by many. He has a great opportunity for achievement and has the general goodwill of the profession and the public behind him.

Correspondence.

POLYARTHRITIS OF UNKNOWN ORIGIN.

SIR: At the suggestion of Dr. Derrick, of the Queensland Institute of Medical Research, I am submitting the following to you for publication.

The disease "known" as polyarthritis of unknown origin aroused interest during the last war. Until recently I was practising in Emerald, Central Queensland, and there I came into contact with at least five (5) cases during April and May of this year.

Clinically this disease consists of a mild arthritis, particularly of the interphalangeal joints, with lymphadenitis, a variable and transient rash, low pyrexia, with usually complete recovery within three weeks, and no complications.

The disease is usually mild, and unless the patients note a rash (which is not always present, and is always transient) they are not likely to consult a doctor. In some cases, however, it leaves a residual stiffness and pain, particularly in the larger joints, that may persist for months.

I was closely associated with the epidemic of this disease in the Northern Territory during the last war. Halliday's account of the epidemic was based largely on patients and information supplied by me whilst stationed as a regimental medical officer in the Adelaide River area. I am therefore well acquainted with the disease. There were a number of cases in the Bougainville campaign, but there the disease never reached epidemic proportions.

The method of transmission of the disease is unknown. Some comparisons and reflections on these might help towards a solution.

Emerald is about 150 miles west of Rockhampton, about 300 feet above sea level; the country is mainly flat, vegetation is a mixture of small timber and scrub, alternating with big stretches of "downs" or open treeless grasslands; annual rainfall 25 inches. The country therefore is roughly similar to the Adelaide River area. The five cases in Emerald were, however, amongst town people with urban occupations.

The insect life had one similarity of interest—March flies were prevalent at the times of both outbreaks.

The method of spread in the Northern Territory was, however, more suggestive of a droplet infection, several inmates of one tent becoming infected successively. On the other hand, in Emerald, although four of the five cases were connected with railway employment, they were not contacts. Nevertheless from questioning the townspeople it appeared likely that there were actually a considerable number of similar cases who did not report sick. This would certainly have been the case amongst many of the Northern Territory cases had they been civilians.

Dengue fever with its Aëdes mosquito vector was fairly common in both outbreaks. German measles (from which a differential diagnosis is often difficult) was relatively common during both outbreaks. I saw an unusual number of cases of *pityriasis rosea* at Emerald earlier in the year. Chickenpox and herpes were common prior to the outbreak, and a few cases of what I believe were mild scarlatina.

All the cases seen in Emerald were adult, three men and two women—one of whom was five and a half months pregnant.

A history of mild upper respiratory tract infection preceding the attacks was frequent in both series, but not constant.

It will be interesting to hear if further outbreaks of the disease are reported. New reports may throw light on the method of transmission.

Yours, etc.,

B. SHORT.

Lady Davidson Home,
Turramurra,
New South Wales.
October 27, 1949.

QUEENSLAND FEVER IN AUSTRIA.

SIR: In "Current Comment" of October 29 you invite Queenslanders to be indignant at the widespread use of the term "Queensland fever", and I dutifully lash myself into fury to comply.

The name "Queensland fever" is, in fact, historically incorrect. The "Q" of "Q fever" is derived from "Query", not from "Queensland". Before this name was chosen in

1937, some other names had been proposed. "Abattoir fever" was rejected because of a possible implication of danger in the meat supply, and also because cases were occurring among dairy farmers as well as among meat workers. The name "Queensland rickettsiosis" was considered, but rejected, because it was thought that the disease might later be found outside Queensland—perhaps over the border in northern New South Wales. There was no suspicion then of a world-wide distribution.

It is comforting to learn from "Current Comment" that "Queensland" is now being contracted to "Q", thus completing the circle and arriving again at the starting point.

Finally, sir, have you considered the predicament of those countries whose alphabets do not possess a "Q"? I believe the Cyrillic alphabets are such. Unless their savants are ingenious they may never be able to have "Q" fever.

Yours, etc.,

E. H. DERRICK,
Deputy Director.

The Queensland Institute of Medical Research,
Herston Road,
Valley,
Brisbane.
November 4, 1949.

THE FEDERAL MEDICAL WAR RELIEF FUND.

THE following contributions to the Federal Medical War Relief Fund have been received:

New South Wales.

A. J. Murray (second contribution), K. W. Starr, £26 5s.

Geoffrey Fenton, E. O. Pockley (second contribution), £20.

A. C. Armstrong, C. A. Holme, S. L. Spencer, R. B. C. Stevenson (second contribution), Weeks White (second contribution), £10 10s.

S. G. Bradfield (second contribution), Helen M. Taylor (second contribution), £10.

N. D. Barton (second contribution), W. L. Kirkwood (second contribution), T. B. Law, N. H. Meacle, £5 5s.

Sylvia D. Bray (third contribution), £5.

H. O. Chapman (second contribution), £4 4s.

M. Andrew, Sylvia D. Bray (second contribution), R. B. Perkins, R. B. Perkins (second contribution), £3 3s.

A. S. Brett, M. E. H. Elliott (second contribution), £2 2s.

Total: £212.

South Australia.

C. Yeatman, £21.

J. E. Bateman, H. H. Formby, R. L. Verco, £10 10s.

M. Mocatta, H. Powell, F. L. Wall and A. W. Wall, £10.

J. L. Dunstone, M. Ericksen, O. W. Firewin, D. G. McKay, F. Boyd Turner, G. R. West, £5 5s.

K. J. Basedow, A. J. Chandler, E. F. Gartrell, R. H. Hamilton, C. T. Piper, H. E. Pellew, W. A. Russell, £5.

M. G. Jansen, A. Britten Jones, £3 3s.

K. F. Cooper, R. H. Hamilton (second contribution), D. G. McKay (second contribution), R. S. Wilkinson, £2 2s.

E. P. Cherry, J. F. Frayne, D. L. Wilhelm, £1 1s.

Total: £166 17s.

Victoria.

D. G. Stewart, £25.

W. L. Colquhoun, K. O'Day, J. O. Laverack, £10.

J. A. O'Brien, G. E. M. Scott, Walter Simmons, £5 5s.

F. J. Colahan, L. Johnston, £4 4s.

G. E. Cole, £3 3s.

Total: £82 6s.

Western Australia.

J. R. Donaldson, £10 10s.

Total: £10 10s.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 29, 1949.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	4(3)	..	..	*	..	..	..	4
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	2(2)	..	..	..	1	..	..	..	3
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(a)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	2(1)	..	..	..	..	..	2
Diphtheria	12(8)	3(2)	4(2)	..	3(3)	..	..	..	22
Dysentery(b)	*	2(2)	..	..	..	..	..	..	2
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	3(2)	*	*	..	..	3
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	1(1)	*	..	1
Influenza	*	*	*	..	*	*	..	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(c)	*	(c)	(c)	(c)	(c)	(c)	(c)	(c)	(c)
Measles	*	*	*	91(48)	*	*	..	..	91
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	9(2)	7(2)	..	21(19)	..	2(2)	..	..	39
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	1(1)	..	..	1(1)	..	..	..	..	2
Rubella(h)	*	*	5(3)	*	11	*	..	..	16
Scarlet Fever	27(16)	13(7)	6(2)	6(6)	3(3)	5(3)	..	..	60
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	..	*	*	*	*	*	..
Tuberculosis(d)	28(23)	13(9)	10(9)	6(5)	15(8)	3(1)	..	..	75
Typhoid Fever(e)	..	..	..	1	..	..	..	..	1
Typhus (Endemic)(f)	..	..	1	..	..	..	..	..	1
Undulant Fever	..	1	..	1(1)	..	..	..	..	2
Weil's Disease(g)	*	*	..	*	*	*	*	*	..
Whooping Cough	*	*	*	32(18)	*	*	..	..	32
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 36, 1944–1945. Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from Northern Territory.

* Not notifiable.

(a) Includes "Mossman" and "Sarina" fevers. (b) Includes amœbic and bacillary. (c) Statistics inexact with varying practice with regard to relapses in Service cases infected overseas. (d) Includes all forms except in Northern Territory, where only pulmonary tuberculosis is notifiable. (e) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (f) Cases reported include scrub, murine and tick typhus. (g) Includes leptospiroses, Weil's and para-Weil's disease. (h) Notifiable disease in Queensland in females aged over fourteen years.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

COURSE IN ADVANCED MEDICINE.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a course in advanced medicine suitable for M.R.A.C.P. candidates will be conducted for a period of fifteen weeks from January 9 to April 21, 1950. Fees for attendance will be £39 7s. 6d. or £2 12s. 6d. per week. The programme has been arranged to take place almost exclusively in the afternoons from approximately 2 p.m. to 5 p.m. on five days per week and will include: didactic lectures on the more obscure aspects of internal medicine, designed to supplement the students' reading, and covering the various systems in turn; lectures and tutorials in electrocardiography; ward rounds and demonstrations of cases at the principal metropolitan hospitals approximately twice weekly; regular clinico-pathological conferences; demonstrations of the *fundus oculi*; lecture-demonstrations in physiology and biochemistry and discussions on applied physiology; lecture-demonstrations in pathology and hæmatology; demonstrations of the application of radiological methods of diagnosis to medical diseases and demonstrations of psychiatric cases.

The supervisor of the course will conduct tutorials on selected subjects and students may discuss with him any problems arising in the course of their work. It is expected that candidates will devote a considerable time to general reading of both text-books and current medical literature. The object of the course is to provide assistance and guidance for the serious students of internal medicine. It is desirable that students should have had considerable clinical experience in hospital and/or in medical practice before considering themselves prepared to take examinations for higher medical degrees or diplomas.

Fees are payable in advance at enrolment date and applications to attend whole or portion of this course should be in the hands of the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, as early as possible.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Thorburn, Campbell Hastings, M.B., B.S., 1944 (Univ. Sydney), 64 Raglan Street, Manly.

Obituary.

MARK CLAYSON GARDNER.

WE regret to announce the death of Dr. Mark Clayson Gardner, which occurred on November 2, 1949, at Melbourne.

Medical Appointments.

Dr. L. M. Archibald has been appointed Medical Officer, School Health Services, Department of Health and Home Affairs, Queensland, in pursuance of the provisions of *The Public Service Acts*, 1922 *to* 1948, of Queensland.

Dr. E. J. Reye has been appointed Medical Officer, Department of Health and Home Affairs, Queensland, in pursuance of the provisions of *The Public Service Acts*, 1922 *to* 1948, of Queensland.

Dr. D. G. R. Vickery and Dr. S. G. Bradfield have been appointed honorary physicians at the Royal Alexandra Hospital for Children, Camperdown.

Dr. D. S. Stuckey and Dr. S. E. J. Robertson have been appointed honorary assistant physicians at the Royal Alexandra Hospital for Children, Camperdown.

Dr. Joan P. Tom and Dr. B. T. Dowd have been appointed honorary relieving assistant physicians at the Royal Alexandra Hospital for Children, Camperdown.

Dr. J. K. Maddox has been appointed honorary consultant to the congenital heart disease clinic at the Royal Alexandra Hospital for Children, Camperdown.

Dr. Guiseppe Pasquarelli has been appointed a quarantine officer under the *Quarantine Act*, 1908-1947.

Diary for the Month.

Nov. 22.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 23.—Victorian Branch, B.M.A.: Council Meeting.
Nov. 24.—South Australian Branch, B.M.A.: Clinical Meeting.
Nov. 24.—New South Wales Branch, B.M.A.: Branch Meeting.
Nov. 25.—Queensland Branch, B.M.A.: Council Meeting.
Dec. 1.—New South Wales Branch, B.M.A.: Special Groups Committee.
Dec. 1.—New South Wales Branch, B.M.A.: Clinical Meeting.
Dec. 1.—South Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester United Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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